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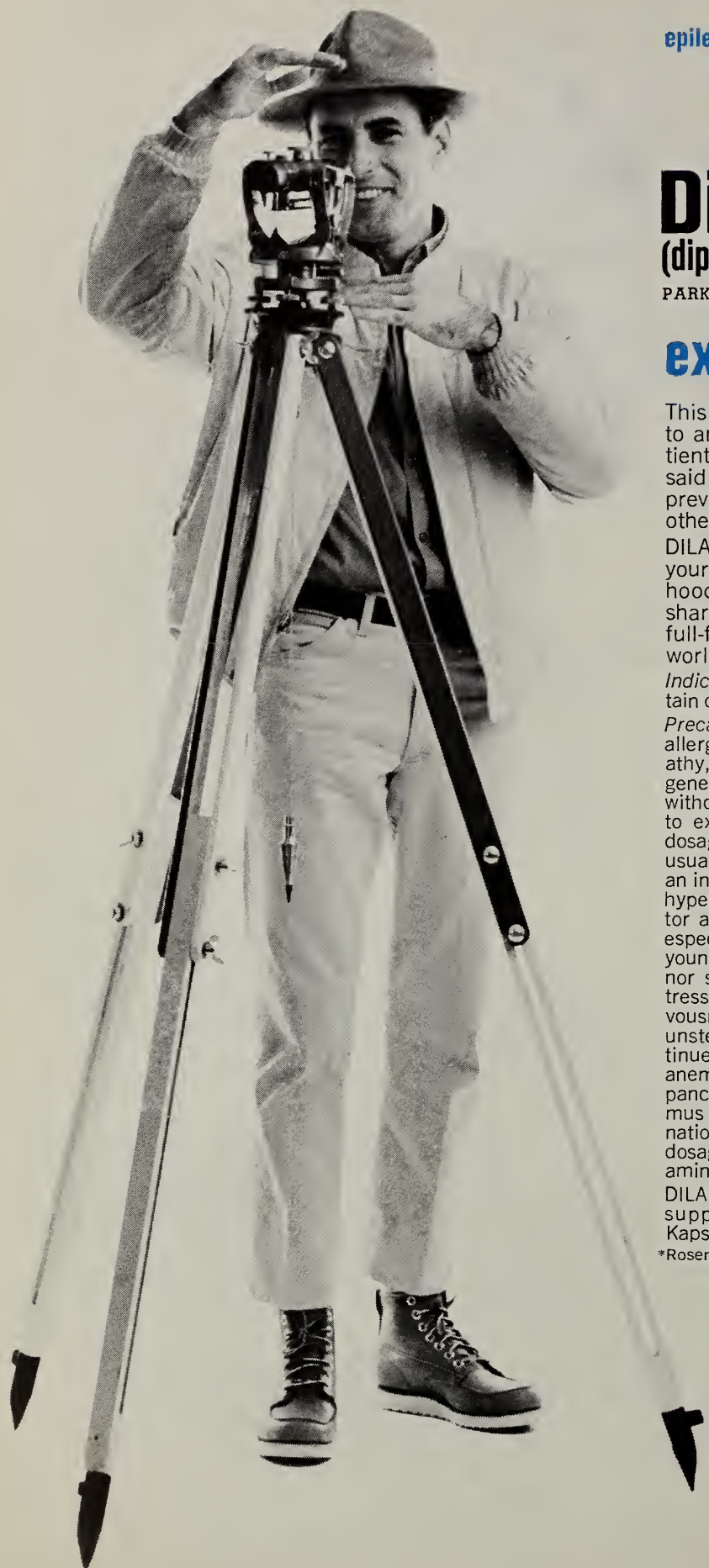
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*Roseman, E.: *Neurology* **11**:912, 1961. 33664

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Office Management of the Emotionally Disturbed Patient

Richard A. Levy, M.D.*

The emotionally disturbed patient often presents a puzzling and frustrating diagnostic and treatment problem to the medical practitioner. Many questions arise when confronted with such a patient: Is the patient psychiatrically ill or am I missing something organic? Should I be treating the patient's depression or his anxiety? Is the patient psychotic or neurotic, and does it make any difference? How should I treat the patient? What drug? What dosage schedule? Would a fifteen minute talk every week do the job? Should I treat the patient? Is commitment preferable? Call in a psychiatrist?

Practical questions, often complex, rarely easy to answer at the moment on the spot. As a psychiatrist, working with many general practitioners, internists, surgeons, and OB-GYN men, I am convinced that they can treat a large number of these patients and do the job well. Let us consider the practical aspects of diagnosis and management.

Diagnosis

As in any other medical problem, a successful approach to the patient is largely based on the initial diagnostic work-up. A very adequate evaluation of the psychiatric patient can be achieved in forty-five minutes to one hour. (Subsequent treatment sessions can be considerably more brief as will be described below.) A concise but thorough history of the development of the patient's psychiatric difficulty is most important. For example, the history of chronicity will help the physician to establish realistic treatment goals for the patient and for the family. A consideration of the patient's history will also give the physician a better idea as to the illness behind the symptoms presented. For example, a patient presenting with depression may be suffering a simple neurotic reactive depression. On the other hand, this symptom may be the only presenting indication of what turns out, through history, to be a slowly insidiously developing schizophrenia. Once again, the treatment approach, the goals set by the physician, and the expectations of the family are all considerably altered by the specific nature of the illness underlying the presenting symptom. An illness which has developed acutely is likely to respond quickly to rapid and judicious treatment. An illness which has been slow in this development and in an individual where there have been repeated episodes of psychiatric disorder indicates the likelihood of a less rapid response to treatment.

History

History material is particularly important in the potentially suicidal patient. When a patient has attempted suicide many times in the past with feelings or symptoms similar to those presenting to the examining physician, it is not likely that there is acute danger of suicide in the current episode. The nature of the patient's past suicidal attempts or gestures, together with his fantasy at the present as to how he will commit suicide is also important in prognosticating the actual chance of successful suicide. For example, an individual who, on many occasions, has swallowed relatively non-toxic medication or who has superficially slashed his wrists is most likely, in the future, to attempt suicide in the same way. He has indicated his wish to remain alive and it is likely that this will continue. Once again, however, it is important for the physician to be certain that there has been no change in the nature of the patient's illness and that it has not progressed into more malignant form.

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This can be done through history obtained from both the patient and the family, ascertaining the similarities in differences between past episodes and the present illness.

Mental Status Examination

An organized review of the patient's mental functioning can be accomplished in ten minutes. The areas that must be investigated are: the patient's general manner and behavior during the examination, the integrity of his thinking processes, his mood, the content of his thinking, sensorium, and judgment. When the mental status examination is approached in a routine and standardized fashion, a great deal of significant information is obtained in a short period of time. Let us consider some examples:

General Appearance and Manner — The patient's dress and attitude often strikingly reveal depression. There is a lack of concern with personal grooming, the patient speaks in a heavy, depressed and labored tone. Movement is made with great effort and the patient's facial expression is one of great despair. Nevertheless, the patient is in good contact with the interview situation, is well aware of his surroundings and will respond appropriately to the interview situation. The psychotic or schizophrenic individual may also show a disregard for his personal appearance. However, depression is not the outstanding feature and the patient is not in good contact with his surroundings nor are his responses always appropriate to the situation. Movement and facial expression are not reflective of deep depression but rather of a dullness, an almost affectless state. There is apathy rather than depression. It is during this stage of the mental status examination that the physician's intuition comes into important play. In sizing up the patient from general appearance and manner, the physician usually arrives at a tentative feeling or diagnosis. Much of what follows in the mental status is essentially an attempt to verify this original impression and rule out the various alternatives.

Evaluation of Mental Processes — Often a patient will appear confused and not make sense to the examiner. The physician will ask himself, "what did he mean by that, I don't seem to catch his drift or understand what he is trying to get across." This will happen frequently enough so that the physician becomes aware that this is a matter of confused communication on the part of the patient. This is typical of the psychotic illnesses and rarely so in simple depression. Also characteristic of schizophrenia, for example, is the inability to think abstractly. This can be evaluated from the patient's general trend of thought and can be more specifically and quickly measured by presenting the patient with a variety of proverbs and asking him to interpret. The classical response of the individual defective in abstract reasoning to the proverb, 'people in glass houses shouldn't throw stones' is, "because they will break the windows."

Mood — This area of the mental status is investigated for the purpose of determining pathology in the realm of affect. That problem presenting most frequently is depression. The patient will reveal such a mood disorder through his general attitude and behavior as described above. Most important are the alterations in daily functions which are classically brought about in depression, namely, appetite, sleeping, sexual drive, and general psychomotor activity. The typically depressed patient tells you that he has progressive difficulty in sleeping, either being unable to fall asleep or waking up early in the morning. His appetite is poor and he has usually lost several pounds. There has been a lack or decrease in the amount of sexual drive or a total loss of interest in sexuality. General activity is greatly decreased and the patient states that he feels sluggish, moves slowly, and has lost all his get up and go. Thinking is described as having slowed down and the ability to concentrate and give full attention to the task at hand is also impaired. The patient will also mention his inability to read a book or watch a television program for any length of time. The patient's thoughts will usually return to his preoccupations. Many depressed patient's will describe feelings of guilt and self-accusation. They tend to heap blame upon themselves. An important practical area to be assessed in any depressed patient is that of self-destructive rumination. It is most important to enter this area in the interview. Many physicians hesitate to open up this area of discussion in the feeling that they would perhaps suggest this to the patient or increase the danger of suicide. This is not true. If the thought or desire or need for self-destruction is an active danger, you can be sure that it was there before the patient ever walked into your office. It is also important to give the patient the feeling that none of the things that he feels or experiences are so terrible that even his physician cannot discuss them or broach them for conversation. Often when you ask a patient "do you feel like committing suicide," the patient will say no. It is important not to rush ahead to the next topic but rather make sure that self-destructive ideas are not present. If the question is asked "does life seem so difficult that you would rather not be here," the response is often "yes, Doctor, I sometimes wish I were dead." This, to the patient, does not mean he wants to commit suicide and yet to the physician is an indication of suicidal potential. It is then important to proceed and ask if he has ever thought of some way of bringing about his leaving the world or in ending his life. If the patient has a well-formed plan then the potential for suicidal acting-out is considerably greater than in the depressed individual who only has vague ideas in this direction. (The exception to this is in the psychotic individual who, although vague in his self-destructive rumination, may act out spontaneously and impulsively.) The nature of the patient's suicidal rumination is also an important indication not only of the type of illness (neurotic depression or psychosis) but also is an indica-

tion of the likelihood for successful suicidal acting out. For example, the individual who expresses a plan of self-destruction via hanging, fire, or some particularly unusual method is most likely to be psychotically depressed and a much greater risk. The individual who talks of suicide via taking pills or cutting their wrists, or turning on the gas, are usually less disturbed. This does not mean that their potential for committing suicide is to be overlooked but rather that the physician can be more relaxed in beginning the patient on a treatment regimen, knowing that the patient is being supervised by the family and is unlikely to try some sudden, impulsive or bizarre method of self-destruction.

The other disorders of affect, manic-depressive psychoses, and cyclothymic personality disorders are quite complex and require the evalution of a psychiatrist. This is particularly true of the adolescent who presents with intense mood swings, failing function in school and socially, manifests withdrawal and leads one to the differential diagnosis of adolescent adjustment reaction or underlying schizophrenia. These individuals should be rapidly evaluated by a psychiatrist experienced in dealing with adolescents.

Content of Thought — It is through the investigation of this area that the physician learns the specific nature of the conflicts facing the patient. It is important to allow the patient a few minutes at the beginning of the interview for free and spontaneous discussion of those problems most concerning him. In addition to the patient's general expression of his feelings and attitudes, we are also concerned with the presence of delusions or hallucinations and their nature. Once again, these areas should be specifically investigated and not avoided for fear of bringing such psychopathology about. When the question "do you hear voices or see things that are not there" is asked, the patient will often say no (in the presence of hallucinations) because of his association of such a question to "being crazy." Therefore, if the question is phrased in a roundabout fashion such as "when you are in a room by yourself, do you have the idea or do you hear your name being called? Or do you get the feeling that someone is outside the room talking about you or making noises?" In the most sensitive patients one can often approach this area by asking "do you have any difficulty with your ears or hearing" and gradually work up to the specific area of hallucinations. Delusions are most often approachable by asking the question "do you ever have the feeling that you are being treated unfairly or being picked on." This question, specifically designed to uncover paranoid ideation, will often lead to the direct delusion of being "spied on by the F.B.I." The importance of uncovering such material is exemplified by the condition of paranoid schizophrenia, where the patient may present a very integrated facade during the initial interview. If the area of paranoid delusion is not entered upon, one might have the impression that the patient is entirely normal and it is difficult to understand why he is in your office. As the paranoid area is opened up, and often this is only one fixed delusion (e.g. being the victim of a Communist plot) it rapidly becomes apparent how the patient's delusional thinking interferes with and distorts his life. The content of psychotic material will, of course, have important bearing on the eventual treatment.

Sensorium — This next area of the Mental Status has to do with the patient's orientation. Confusion as to place, person, and/or time indicates the presence of severe psychological disease or organic brain impairment. Patients may appear deceptively well oriented. One might be surprised to ask his patient where he is at the moment of examination and receive a most unusual and incorrect answer. This is an area of the Mental Status which must not be assumed but which definitely should be gone into. This is particularly so in view of it being one of the primary mental symptoms of organic brain disease. Included with this particular section of the Mental Status should be an evaluation of the patient's intellectual abilities. This area, when impaired, is also an indication not only of emotional disease but often of organic brain disease. Not only can history, in terms of changing intellectual function, afford opportunity for good evaluation of intellectual function but there are certain simple and rapid interview techniques which may also be utilized. For example, the patient is asked to subtract seven from one hundred serially, taking seven from each remaining amount. Psychiatrists and neurologists working with brain damaged individuals claim that such disease will always be revealed by this particular test even though it might require completely running down the series from one hundred to zero. Depressed patients will often give the history of being unable to concentrate in reading or watching television. They will also state that they "do not have a head" for figures and are having increasing difficulty with family finances and budget. Often a patient's awareness of reality as well as his intellectual capacities can be estimated through a brief discussion of some particularly significant current event. The patient's ability to reason abstractly, his contact with his surroundings, and his intellectual capabilities can all be measured within a short period of time.

Judgment — The next section of the Mental Status is once again particularly important in ruling out organic brain disease. The majority of patients with psychiatric illnesses will suffer some impairment of their judgment; however, those defects revealed by the brain-damaged individual or the patient with 'organic brain syndrome' will show striking abberations in this area. A simple test other than a general impression of the patient's judgment through conversation concerning his problems and thoughts for the future is to present him with a test situation, e.g. "what would you do if you found an envelope, sealed, addressed, and stamped, lying in the street." Most patients will say, "put it in a mail box." The patient with organically based emotional disease might answer, "I would rip open the envelope

and read the letter because I would be curious to see what it says" or "I would put it in my back pocket." A classic question asked to determine state of judgment is "if you were in a movie theatre and someone yelled 'fire,' what would you do." The degrees of impairment, of course, can be evaluated from the patient's response. These test situations are short cuts but, in my opinion, not as effective as estimating the patient's judgment from those events which are current in his life and in which he has expressed his opinion. For example, it is very important for the physician to know whether the patient is fully aware or sufficiently aware of his illness and its consequent limiting influences. It is also important to know, in the depressed patient, whether or not they accurately judge their suicidal tendencies and would be able to call for help when necessary. The physician must know how intact the patient's judgment is in terms of appraising the family of their role of supervising the patient and cooperating with treatment. There are also the legal implications of an individual whose judgment is impaired by mental illness be it functionally or organically.

This completes the various sections of the Mental Status Examination and should give the physician sufficient information to enable him to formulate a practical approach to the patient's problem. A good portion of the Mental Status Examination was actually completed during history taking and physical examination. Those areas which are not completed may be so done following the physical. This is a particularly good time to enter upon areas sensitive to the patient in view of the likelihood of increased confidence and comfort with the interview situation.

Physical Examination

There is one unfortunate aspect of the publicity mental illness and emotional disease has received in recent years. This has been the too rapid acceptance of the symptoms of mental illness on a functional basis and the neglect of adequate examination to rule out possible organic etiology. The patient with an apparent psychiatric problem should be given the benefit of at least a minimally adequate physical examination including a gross neurological. This will help to avoid the now frequent use of the waste-basket psychiatric label.

There is, occasionally, a reluctance on the part of physicians, psychiatrists not excluded, to perform physical examinations on an emotionally disturbed patient. The basis for this reluctance is centered about the concern that the patient will interpret such an examination as a physical assault or as a hostile act on the part of the physician and his nurse. Associated with this concern is the question "how will my patient be able to talk to me if he looks upon me as someone who manhandles him rather than an understanding and gentle friend?" After having performed many physicals on patients admitted to the Acute Admission Services of state, general and military hospitals, I am convinced that the physical examination is helpful in establishing the patient-doctor relationship in the emotionally disturbed patient as well as in any other patient. The routine physical examination is a familiar landmark to most individuals. The white coat, the stethoscope, the sphygmomanometer, and the nurse, frequently offer an island of security and familiarity in the midst of a frightening sea of mental confusion. This principle applies to the less disturbed patient that is likely to be seen in the general practitioner's office as well as the more severely disturbed patients admitted to Acute Psychiatric Hospital Services. I would urge the medical practitioner to include the physical examination in the initial part of his approach to the emotionally disturbed patient.

Treatment

The double approach of psychiatric drugs together with psychological exploration and understanding yields gratifying and often rapid results.

Psychological Approach

A psychological approach does not require deep psychoanalytic exploration, nor are hours of searching and puzzling necessary. As a physician you are particularly well trained through education and experience to recognize emotion and its physiological components. Helping the patient to be aware of an emotion, e.g. anger, through recognizing the subjective feeling and the bodily expression of this feeling can bring about dramatic results. Tying the recognized emotion to a real life situation is the next step and with repetition of this cycle, a therapeutic and helpful approach has been initiated. Many individuals become emotionally ill because they do not know or recognize what they feel and when they feel it; the emotion becomes distorted and twisted in its eventual coming to light. For example, the patient who is reasonably angry but is unable to so recognize and direct such feeling presents with a depression and suicidal rumination (and no apparent evidence of anger). Once again, deep delving into the unconscious is not necessary. The direct psychological approach to this patient is to help him become aware of his anger rather than to tell him of it. For example, certain individuals when unable to find adequate release for a repressed emotion develop headache. A physician may conclude from his patient's history that headache is usually developed under certain situations in the home. Physician can help the patient to associate headache with the repetition of certain situations irritating and frustrating to the patient and eventually the patient will be able to realize when he becomes frustrated and angry. The next step is to help the patient more directly express the feeling underlying headache in an appropriate manner and at the appropriate time. It will sometimes be necessary for the physician to say to his patient, "you always seem to develop a sinus headache when your wife leaves Friday

evenings to play cards with her girl friends. I wonder if there isn't something about that situation that bothers you." At first the patient will be unable to clearly and concisely phrase his feelings and it will require some patience on the part of the physician to help the patient to become aware of what he feels, albeit in some vague and diffuse way, when his wife leaves. It may eventually come to light that the patient feels this is the only time of the week when he has any opportunity to discuss personal family matters or at the end of a long hard week, get many things off his chest that have been building up. It may take one entire half hour visit to help the patient realize how he is upset or disturbed when his wife leaves him, but the rewards of such recognition in terms of future conflict situations are great. At the same time the physician is establishing a pattern for the patient for not only the next talk but in his life at home.

It is often difficult for the non-psychiatrist physician to map out a treatment plan along psychological lines for the emotionally disturbed patient. Being inexperienced in the technical aspects of this approach he is used to 'flying by the seat of his pants' and is hence uncertain of his ground. Psychiatric consultation at the very beginning of a case can be most helpful in outlining such a treatment plan. One or at the most two consultations with a psychiatrist should be quite adequate in developing a psychological plan of approach for the general practitioner. Most psychiatrists, particularly in this area of the country, have full case loads and long waiting lists. They would be pleased to have a referring physician request a psychological treatment approach to the patient. There has never been sufficient psychiatric treatment time available for all those patients requiring such help and there never will be. Physicians have become resigned to the fact that many of their patients requiring psychiatric help will never get to the psychiatrist or at best, will get to one after many weeks of waiting. They have become used to sending patients for single-shot diagnostic consultations and have expressed the feeling, following such consultations, that now they know exactly what they knew before they sent the patient to the psychiatrist. Rather than ask simply for a diagnosis and disposition, I would suggest that the referring physician express an interest in treating this patient with drugs and a superficial psychological approach as recommended by the consultant psychiatrist.

Drugs

Psychiatric drugs are quite helpful in directly treating presenting symptoms as well as increasing the patient's potential for a supportive psychotherapeutic approach. These drugs are not multi-purpose, but have been formulated for specific symptoms. For example, Chloropromazine (Thorazine®) is primarily designed for and has been most useful in treating acute psychoses and nonspecific intense anxiety. Depression is often intensified and aggravated by Chloropromazine and a patient whose outstanding complaint is depression should not be treated with this drug. This is a specific action of a psychiatric drug that is not widely appreciated and is a good example of why the physician who wishes to treat emotionally disturbed patients should be quite familiar with the specific uses of the wide gamut of psychiatric medication. The most reliable of up-to-date information concerning these drugs is a current psychiatric journal, particularly the *Journal of the American Psychiatric Association.* Very few of the many drugs developed have produced consistent and reliable results. High powered advertising campaigns and glib salesmen, loaded down with samples, are not reliable and often lead to drug misuse and patient abuse. Postgraduate clinical seminars in the office management of psychiatric problems are practically oriented and will give the non-psychiatrist physician a reliable and tested approach to their patient's illness. (A clinical seminar of this nature was conducted by the Bureau of Mental Health in Augusta during the Spring of 1964 and an on-going continuous practical case seminar is being held at the Maine Medical Center, Mental Health Clinic.) A brief call to the local Mental Health Clinic or psychiatrist will give you the latest information on the latest psychiatric drugs. Before going on to specific psychiatric illnesses and their treatment, the phenomenon of 'placebo' response to medication must be discussed. We are often pleased at the initial response to a medication only to find that within two or three weeks or sometimes longer, the old symptoms and problems have once again returned. It becomes apparent, particularly in certain patients, that they are likely to respond favorably to just about anything that is done for a short period of time. However, there are specific implications for the emotionally disturbed patient and his treatment. One of the great difficulties in treating such a patient is arriving at an adequate dosage. Rather than become discouraged at this placebo response, it is important to remain with one reliable drug and increase the dosage to an adequate amount over a sufficient period of time before giving up and moving onto the next drug. For example, Tofranil® is an anti-depressant medication which may require as much as thirty days of continuous therapy at high levels before a satisfactory result is obtained. The administration of 75 mg. daily of this drug for a period of two weeks may not produce a beneficial effect whereas the dosage of 150 mg. daily may startlingly alleviate depression. The cardinal rule for the drug treatment for the emotionally disturbed patient is The Proper Medication In The Proper Dosage For The Necessary Period Of Time. Let us consider the specific types of illness that a patient is likely to present with at the office and their treatment.

Common Psychiatric Disorders And Office Treatment

Although it is unusual for a patient to come in with one clear-cut specific symptom, there usually being a

mixture of many, one symptom or complaint will usually be emphasized. Frequently, the practical problem of disposition will take precedence and treatment thereby regulated. For example, the patient who presents with anxiety, agitation, depression and acute suicidal ideation, presents primarily a disposition problem. One must be certain that any treatment initiated can be properly supervised by the family and that the patient can be adequately supervised in the home situation. Once disposition can be settled, depression, the outstanding and most dangerous symptom in this patient, must be treated as the primary target. This primary goal treatment approach can be supplemented by the combined use of drugs. Examples will be given below.

Depression

Drug Treatment — The most commonly used and reliable anti-depressants are Elavil,® Tofranil, and the MAO inhibitors. The newer anti-depressant medications are theoretically designed to act at the point of biochemical aberration causing the symptoms of depression. They are, therefore, quite different from drugs such as Dexamyl® which are similar to the use of Aspirin for headache without attempting to treat the etiology of the headache. Regardless of the psychological conflicts bringing about depression, it is now widely accepted that such conflicts are mediated through biochemical processes and hence these drugs are aimed at restoring the biochemical aberration or imbalance. Ritalin® has produced inconsistent results and I have found, not helpful in cases of serious depression. It could be categorized as a mild psychic energizer. Let us consider each of the drugs mentioned individually as to their specific characteristics and dosage. Elavil is one of the most effective, if not the most effective, anti-depressant drug in cases of acute depression. When prescribed in a beginning dose of 25 mg. t.i.d., one can expect to see positive results within three to seven days if there are going to be positive results at all. The patients will usually experience considerable lethargy during the first three or four days of this therapy before such effects wear off and the patient becomes more alert. I have found this drug to be particularly effective in patients complaining of severe insomnia with feelings of depression. Appetite is usually promptly restored and the patient begins to gain weight rapidly. If there is no positive response within one week and there have been no sensitivity reactions to the drug, I will immediately increase the dosage to 150 mg. daily. I often find that the minimal therapeutic dosages recommended are not really sufficient to bring about the desired anti-depressant effect and that the test dosage of 25 mg. t.i.d. (and this is certainly true of the majority of anti-depressant medication, i.e. recommended beginning therapeutic dosage is not sufficient to produce maximal therapeutic effect). A starting minimal therapeutic dosage of Elavil then in a patient who is truly depressed is 25 mg. t.i.d. It is also helpful in the treatment of depressed patients to help them directly with their sleep problem by prescribing a mild hypnotic such as Chloralhydrate. If there has been no adequate response after five days of treatment with Elavil, I would elevate the dose to 150 mg. daily (50 mg. t.i.d. with meals) and wait one more week before discontinuing the drug. If there is still no further response I would wait forty-eight hours prior to beginning any new anti-depressant. This latter period of waiting between drugs is an advisable rule to follow. There have been some unfortunate incidences involving the overlapping use of medications with great discomfort or precipitous depressions resulting. There are a variety of side effects using Elavil, as there are in almost all of the various tranquilizing and anti-depressant medications. They are usually tolerable, certainly so when stacked up against the potential benefits of the drug. I have found it most helpful to inform the patient and his family of what they may be, thereby avoiding considerable anxiety and frequent phone calls. The most common side effect complained of is lightheadedness due to the hypotensive effect but particularly so when suddenly lifting one's head or changing from the sitting or standing position. This is usually accommodated to and certainly can be tolerated during the treatment. I do not find that advising patients and families of possible side effects increases through suggestion the incidence of such effects. Tofranil is more helpful in depressions of long standing. When the problem is not as acute one can also afford to wait longer for the anticipated relief. The beginning dosage is the same as Elavil but the expected periods of response should not be looked for in less than two weeks. I have found that if there is going to be a marked response in the future, that there is usually some indication in this direction during the first two weeks. Thirty days may be required before seeing a response with this drug. I have once again found it helpful, after a period of two weeks if there has not been a gratifying and satisfactory response prior to that time, to increase the dosage to 50 mg. t.i.d. If there is no effect within six weeks I feel the drug has been fairly tested and should be discontinued. There are a variety of minor side effects reported by patients taking this drug which are thoroughly described in the literature put out by its manufacturer. None of them have been serious enough, in my use of the medication, to warrant its being discontinued. The MAO inhibitors as a group have produced good results in individual cases. However, on the whole, I find them comparatively unreliable and inconsistent in their therapeutic effect. Psychiatric literature is filled with many contradictory papers dealing with these particular drugs. Their side effects are often particularly unpleasant and cannot be tolerated by the patient. I personally use them only when I have found no adequate response in the use of the two previously mentioned drugs. It should be emphasized that this is my own personal experience with this particular group of drugs and should not discourage any physician who has had good success with

them and is well acquainted with their dosage schedules as well as side effects and specific case benefits.

It is very important for a physician treating such patients to become familiar with a few tried and tested drugs and stick with them rather than hop around from one medication to another. Good background experience will be obtained and adequate dosage levels achieved.

Electroshock Therapy — with the advent of anti-depressant medications, many psychiatrists, state hospitals, and psychiatric units have abandoned the use of this form of treatment for depression. Nevertheless, there are cases of depression (particularly psychotic depression, e.g. involutional melancholia) that are refractory to medication and respond well to E.S.T. If the patient did not respond to your use of pharmacotherapy, a psychiatric consultation to consider the use of E.S.T. or other forms of treatment is advisable.

Psychological Approach — It was stated earlier in this paper that it was not necessary for the physician to become involved in lengthy discussion sessions in the process of treating the emotionally disturbed individual. Although this is true, it is important that discussions be held regularly and consistently according to a specific plan. There should be a concentration of interviews during the beginning or the crisis of the patient's illness. For example, a usual schedule would be a second follow-up interview following the initial examination in three to five days for the purpose of evaluating the effects of the medication, ascertaining the mental state of the patient and once again, reviewing their supervisory duties with the family. Occasionally it is not necessary to see the patient for a week, having follow-up information phoned in by the family. The second and third interviews should be scheduled a week apart, and if there has been good progress shown by that time or an indication of progress it is then quite appropriate to spread out the interviews at two week intervals. In treating cases of acute depression at the Mental Health Clinic at the Maine Medical Center, we have found that the acute crisis is usually resolved and the patient well on the road to recovery within three to four interviews. Rarely are more than a total of six required before the patient can be put on long-term follow-up status when he would be seen for his seventh interview in approximately one month to six weeks. The aim of this approach to the patient is to resolve the acute crisis, not to revamp or change his personality. We are simply concerned with alleviating the depression and restoring the individual to a functional status. It is to be expected that he may, at some time in the future, have further difficulty. It would be unrealistic on the part of the family physician to expect to be able within the time allotted and within the realm of his technical skill, do anything more than bring about such a resolution. To review briefly, the initial interview should last approximately fifty minutes to one hour and all subsequent interviews no more than thirty minutes. The next step involves the question "now what do I say to the patient?" The depressed patient is often quite difficult to communicate with. Thinking and action are usually slowed down and he therefore will not offer very much spontaneous discussion. The interviewing physician will often feel as if he "were pulling teeth." Nevertheless, depression is caused by some factor or factors that have been eating away at the patient and are related specifically to certain events in the patient's life. Cases of acute depression can usually be traced to some acute precipitating event such as the death of a family member or close friend. It is then necessary to get the patient to talk as much as possible about his feelings concerning the traumatic event. Another common type of depression is that caused by great dissatisfaction, annoyance or anger, again with people close to the patient but which has been unexpressed and continually pushed down. These feelings appear to be tearing away at the individual himself rather than at all those people with whom he is dissatisfied and angry. Once again, it is very important to encourage the patient and allow him to express these feelings more directly. It may be quite frustrating to the interviewing physician to have this come about quite slowly but if the patient is able to express, to any degree, these kinds of feelings within the first two interviews, it is certainly worthwhile and is laying down the pattern of your brief psychotherapy for future sessions. As in any illness, it is quite important to get to the precipitating roots or events and explore with the patient all of his feelings and thoughts in relation to them.

The central goal of the psychological approach with the psychotherapy of depression and essentially all other psychiatric disorders is to help the patient become aware of his feelings with specific relationship to those events occurring in his life. I would like to emphasize that this goal is to be carried out in the direction of the patient's present or recent life, not his long distant past, not through the investigation of dreams, not through dredging up so called 'traumatic childhood experiences.' The goal at hand is to help the patient over his acute psychiatric crisis, not to explore or renovate the deep psychic structure of his personality.

Environmental Approach — It is occasionally recommended that a patient "take a trip" or go to live with some other family member in order to speed recovery from a psychological illness. Unfortunately, the patient takes his problems with him and this, in reality, does no more than temporarily remove the problem from the household and the community. The actual circumstance involving a truly harmful and noxious family situation for a disturbed patient justifying geographical removal is rare. This form of approach to mental illness is not recommended.

Anxiety

Anxiety is usually seen by the medical practitioner in one of two forms. The patient may be frankly anxious, describing a feeling of continual unrest and apprehen-

sion and stating that he feels something terrible is going to happen but is unable to put his finger on the dreaded event. This will involve the subjective symptoms of restlessness, agitation, the inability to concentrate on tasks, and preoccupation with the continual worrying over a menagerie of minutae. The second common form is the expression of anxiety indirectly through a host of somatic symptoms and worries. This type of patient is familiar to the general practitioner and fills the out-patient department of every hospital. There are usually no positive physical findings or physical findings which cannot substantiate or explain the nature of the patient's subjective complaints. Reassurance is short-lived and the absence of positive findings on medical examination is only temporarily reassuring. There are certain typical physical symptoms associated with this problem such as a feeling of tightness in the chest, difficulty in swallowing, tension or pain in the back of the neck usually involved with a variety of headaches, vague gastrointestional complaints and the more classic vague migratory aches and pains of an inconsistent and baffling nature. Unfortunately, these patients often end up as doctor and clinic "shoppers." They present one of the most difficult problems to the psychiatrist as well as to the family physician; however, there is one specific combined approach of both medication and brief superficial therapy which has been helpful to a large number of these patients.

Drugs — There are a variety of preparations available on the market ranging in potency from Librium® through higher dosage levels of Thorazine. Once again, it is important for the physician to become familiar with one or two proven and tested medications. I have found Stelazine,® when used in adequate dosage levels, to be the most effective drug in dealing with such patients. Effective dosage means starting with 2 mg. on a b.i.d. schedule. The patient will usually indicate some relief at this level and if there are no sensitivity reactions nor untoward effects, I will rapidly elevate the dose to 5 mg. hoping to achieve maximal therapeutic effect at this level. Frequently this is not sufficient to help the patients during acute crises of anxiety and I will add Thorazine 50 mg. on a t.i.d. or q.i.d. basis. If lethargy results, I inform the patient that this is likely to wear off in two or three days and is worth the temporary discomfort. This usually happens but if not, Thorazine may be reduced to lower levels. Many physicians feel they have had good success with Librium and its newer cousin, Valium.® I have found Librium to be rarely successful in the more acutely anxious and disturbed patients, particularly during periods of crisis and Valium is in its youth. Meprobamate, a widely used and abused relaxant and tranquilizer, has yielded inconsistent results in cases of severe anxiety. In my opinion, this drug is most helpful when administered in a dose of 400 to 800 mg. h.s. with a patient who is depressed, agitated and having difficulty in falling asleep. Cases of habitution to Meprobamate have been reported as well as convulsions upon withdrawal from excessively large dosage. There are a plethora of other medications touted as being effective in cases of anxiety which I shall not describe. It is my observation that when a physician utilizes one or two tried and tested drugs in adequate dosage that his success rate in helping patients with anxiety as well as other psychiatric disturbances is greater than when switching from one drug to another.

Psychological Approach — Once again, an attempt to find the precipitating events surrounding the patient's anxiety is important to attempt. However, in the majority of patients with this particular problem, it is unlikely that clear-cut precipitating events, or a specific relationship or episode is as likely to be found as in cases of depression. The illness is usually developed in an insidious, vague and diffuse manner. The realistic goal in terms of treating anxiety under the conditions we have outlined above (in the office over a relatively short period of time with brief treatment sessions) is to decrease the intensity of the anxiety, increase the length of time between episodes of panic and help the patient establish a state of tolerance to what is likely to be long-term feelings of anxiety. Let us take the rather classical case of a patient with "cardiac neurosis." This patient usually presents with a variety of somatic complaints initially attributed to coronary artery disease or anginia and eventually, following complete and thorough laboratory work-up, is recognized as having psychological problem.

The first aspect of the combined treatment mentioned above is the administration of an appropriate medication as outlined in the section on drugs. The second step, the psychological approach, is as follows:

"Mrs. X, I know what is troubling you, I have had many patients with your difficulty and I am going to treat you in the proper fashion. First of all I want you to know that you have no real heart disease, that your problem is one of nerves, that you are emotionally upset. Now let us review those times when you have felt this pain or discomfort and have had the fear that you were going to die of heart disease. . . . . It would seem, from our discussion, Mrs. X, that whenever something bothers you or upsets you that you begin to get these feelings" or "Mrs. X. although we cannot find the specific thing that causes you to be upset before you have this difficulty I am sure that we will eventually get to the root of the trouble. Now let us take a look at what happens when you have these attacks — you become upset, you feel like something is wrong with your heart, you experience pain, you become frightened, you are convinced you are going to die. But you never die, do you? You have visited many doctors, have had many laboratory tests, nothing has ever been found and yet you continue to feel ill. Then, Mrs. X, it would appear that we will never find the answer by going from one clinic or one doctor to another and having all these laboratory tests. You have had vitamins, many different kinds of medication and yet you still have these problems. You and I can conclude, Mrs. X, that (1) you are never going to die

from this problem and (2) that there is no physical illness causing the pain that you feel. That does not mean you will not have these feelings in the future. As you take the medication I give you and as we talk, you will find that you will become less anxious and do not have these attacks or episodes as frequent; however, do not expect to be cured overnight. This is going to take some time. Once again, let's remember that none of the things you have worried about so many times have ever come to be, you never have had a heart attack, the pain eventually does go away and the doctors have never been able to find anything wrong with you. I can guarantee, however, that you will worry about these things in the future. . . now, aren't you a worrier, Mrs. X, don't you grab on to things and worry them in circles, becoming upset and yet not solving the problems or those things that you worry about. Tell me about some of the things that concern you and worry you." The foregoing has been in condensed form, a verbal outline of a discussion with this kind of patient. The basic ingredients are presenting to the patient the image of a physician that is secure, confident, insistent and steadfast in his opinion that there is no real physical illness, that he knows what is wrong, that he is going to offer some relief to the patient which will not be miraculous in its development but is sure to come. The other important aspect of such an approach is the realistic reassurance to the patient that, in spite of the worrying, nothing has ever happened. It will be necessary to repeat this formula at virtually every meeting with the patient. For some reason, which has never been adequately explained, this simple, basic, concrete approach, when utilized together with medication to combat the intense anxieties experienced by these patients, has proved extremely helpful.

Other Disorders

The psychotic patient should always be referred to either a psychiatrist or the nearest mental hospital for evaluation and treatment. During the very early stages of acute upset, the family physician is likely to be called and finds himself with the problem of controlling an irrational and often dangerous patient. Prior to referral the adequate use of the appropriate medication can be most helpful in dealing with this problem. For example, 100 to 200 mg. of Thorazine IM every two hours until the patient is calm is an appropriate drug approach. Treatment of patients with psychoses is a tricky and complex affair and should not be attempted in the office of the medical practitioner.

Alcoholism is one of the most frequent and frustrating patient problems presented to the medical practitioner. Treatment success appears to be more related to the personality of the individual physician rather than the formal treatment utilized. I have often heard professionals specializing in this field state that the most effective tested form of treatment is Alcoholics Anonymous. It is most important, in the alcoholic patient, to rule out the presence of some underlying psychiatric illness such as depression. By forming very close long-term relationships, almost friendships, with their alcoholic patients, many physicians have been able to decrease the intensity and frequency of alcoholic bouts. There is no specific psychological approach to this category of patient to be recommended. For those physicians interested there are journals devoted to the study of alcoholism as well as a host of texts.[1]

Psychosomatic diseases,[2] drug addiction,[3] sexual deviation,[4] senile brain disease,[5] and a host of other conditions bordering on or falling within the realm of psychiatric disorders are not within the scope of this paper. The reader is referred to the literature.

Psychiatric Consultation

You can be greatly helped in your handling of emotionally disturbed patients through consultation with a psychiatrist whether it be in the local community or in some distant city. Do not be satisfied with simply a diagnosis and prognosis. Recognize with the psychiatrist the lack of psychiatric treatment available and your need for an appropriate treatment plan in very specific detail. Have it understood with the consulting psychiatrist that it may be necessary for you to occasionally refer the patient for further consultation during rough periods in the patient's illness and a reevaluation of the treatment program. Frequently, telephone conversation is adequate to discuss an urgent problem. Mental health clinics now offer consultation services in many areas. Although treatment time might not be available and you hear of long waiting lists, you usually can have a patient seen within a relatively short period of time for the purposes of obtaining a recommended treatment program in your office.

Summary

Basic principles of diagnosis and treatment of those emotional disorders commonly coming to the attention of medical practitioners are discussed. Depression and anxiety are used as models, and their treatment, both psychological and drug, is outlined. With a planned approach of diagnosis and treatment, as well as timely consultation, the medical practitioner can become an effective physician in the treatment of emotional disorders in his office.

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Bilateral Acoustic Neurinoma Presenting Clinically As A Suprasellar Tumor*

J. Galindo, M.D. and H. S. Baar, M.D.

Although acoustic neurinomas are not uncommon, considerable variance is encountered in their clinical symptomatology. The following case is an example of such variance:

L.S.C. was born March 20, 1937 at full term, following normal pregnancy, and delivery without employment of forceps. The child was cross-eyed at birth, walked at 14 months, and spoke at 3 years. He progressed normally until the age of 5 years when he had mumps, and during this illness he had several convulsions. Mental deterioration apparently had its onset at this time. He attended school for five years, but was unable to learn and remained in the first grade all this time. Until a couple of years prior to 1958 he had been able to do small jobs around the home, such as bringing in the wood, but soon afterwards he began to have weakness in his legs and could not walk properly. In 1958, slowly and progressively, he became totally blind and deaf. He started to walk with a broad base and later his sense of balance became altered to such extent that he was unable to walk across his room and had to crawl on his hands and knees. He made only unintelligible sounds and would lie in his bed, giggling, for hours. In 1959 he was still able to dress himself if clothes were placed beside him, and he was able to feed himself. In September of 1959 the patient was admitted to the Pineland Hospital and Training Center with a diagnosis of idiocy. Physical examination revealed a dull, apathetic, blind, and deaf, white male, with a moderate degree of lordosis and pectus excavatum.

There was papilledema, more marked on the right than on the left, with bilateral secondary optic atrophy. The pupillary reflexes bilaterally were absent to light. Both pupils were small and equal. The abdominal reflexes were equal and the deep tendon reflexes were increased on the left. There was also a positive Babinski on the left. There was bilateral impairment of the acoustic nerve. Laboratory data revealed a normal urine and normal blood count. Evaluation of the patient's mental status on the Vineland Scale was reported as an SMA of 2 years and SMQ of 9. The skull film showed, in the lateral view, the sella tursica abnormally enlarged, measuring 2 x 1.5 cm. There was no decalcification or visible erosion of the clinoid processes Fig. 1A. There was also no abnormal calcification. The cranial sutures appeared slightly separated on the base film, but were not as distinct as on the lateral view. The impression was of intra-sellar tumor, with question of increased intracranial pressure. A right cerebral angiogram in the A. P. view showed the cerebral vessels well and the callosomarginal artery clearly visible, occupying the midline. In the lateral view, the middle cerebral artery showed minimal narrowing that suggested impingement on it by an extrinsic mass. A beltone audiometric evaluation was done with no observable response, therefore it was felt that hearing loss was total. Slowly and progressively, the patient encountered more and more difficulty in walking. Straight leg-raising one year later had become limited to 20 degrees on the right and 10 degrees on the left with no accompanying spasticity. He had rigidity of the hamstring muscles, and was now unable to walk without assistance. His sense of balance had become impaired to the extent that, unsupported, he tended to lean or pitch to the right. He followed a progressive downhill course and, eventually, became bedridden. Physical examination repeated in the year of 1964 again showed small, round, equal, non-reactive pupils to light. The right eye showed lagophthalmos and absent corneal reflex. The conjunctiva appeared abnormally congested and there was right facial paralysis. The limbs now showed spasticity. The deep tendon reflexes were present bilaterally; they were equal and brisk. Jaw jerks were absent. Babinski signs were negative bilaterally. The physical diagnosis was ataxia, apraxia, spastic quadriplegia, right trigeminal anesthesia; intracranial neoplasm, in suprasellar location.

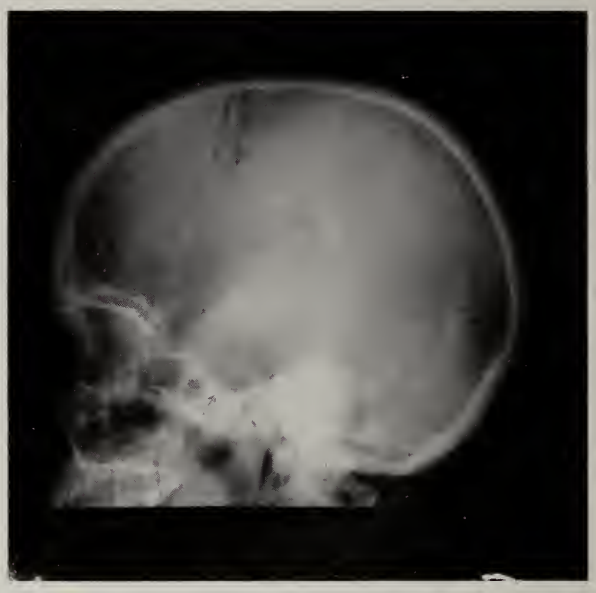


Fig. 1a. The marked enlargement of the sella turcica is clearly evident in this skull x-ray.

*From the Department of Pathology, Pineland Hospital and Training Center.

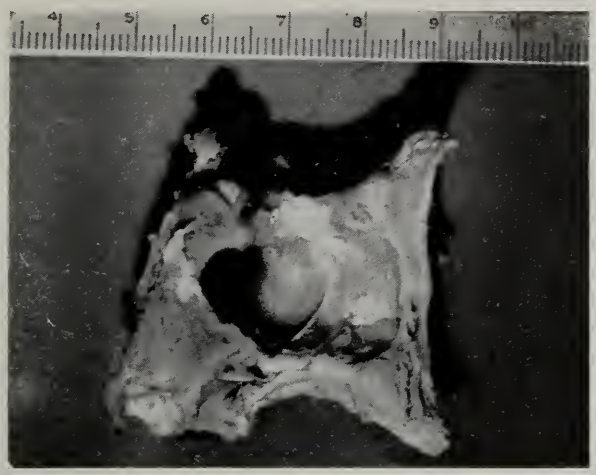


FIG. 1. Enormous dilatation of the sella tursica in all dimensions. A portion of the tenuous diaphragma sellae is evident on the right.
H & E x 160



FIG. 2. Bilateral acoustic neurinomas projecting into the cerebellar hemispheres. The stretched eighth nerve is seen extending through the tumor capsule on one side.
H & E x 160

Over a period of several weeks, the patient deteriorated to the point that he encountered difficulty in mastication and, later, difficulty in swallowing.

A repeat skull x-ray showed an enlarged sella turcica with clearly visible clinoid processes without decalcification and without evidence of absorption displacement or erosion. The sella remained deep, slightly enlarged, and somewhat flattened, and the radiologist then felt that there was no chronic pressure in this area since, if it had been present for long, one would have expected to see absorption of the clinoid processes.

The patient continued to deteriorate; became greatly emaciated, and expired with pneumonia on January 21 of 1964.

Post Mortem

At time of autopsy, the brain weighed 1420 grams; the gyri were flattened and the sulci were narrow. The floor of the third ventricle was visible, projecting downward like a translucent funnel into the sella turcica. The latter was covered by a narrow ring of tenuous diaphragma sellae (Fig. 1). The sella was conspicuous by its large size and depth, measuring 2 x 1.8 x 1 cm. There was no erosion of the clinoid processes and there was no evidence of a suprasellar tumor nor of a pituitary tumor. The pituitary gland itself was rather small, flattened against the sella, it was thin, and covered by a spider web-like membrane.

At the cerebellopontine angle there were two, moderately firm, gray-white, smooth-surfaced, nodular masses 4 cm. x 3.8 cm. x 3 cm. on the left and 4.8 cm. x 3.9 cm. x 2.5 cm. on the right, which were covered by a delicate, translucent capsule in which the eighth nerve was identified, markedly thinned and stretched and embedded within the tissues of the capsule. The trigeminal nerve on the same side was also somewhat elongated and thinned (Fig. 2).

In the anterior portion of each of the cerebellar lobes

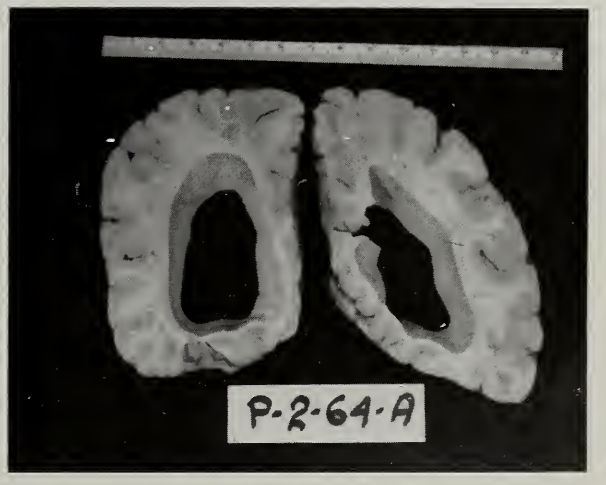


FIG. 3. The lateral ventricles show enormous dilatation as a result of the Sylvian acqueduct obstruction.
H & E x 160

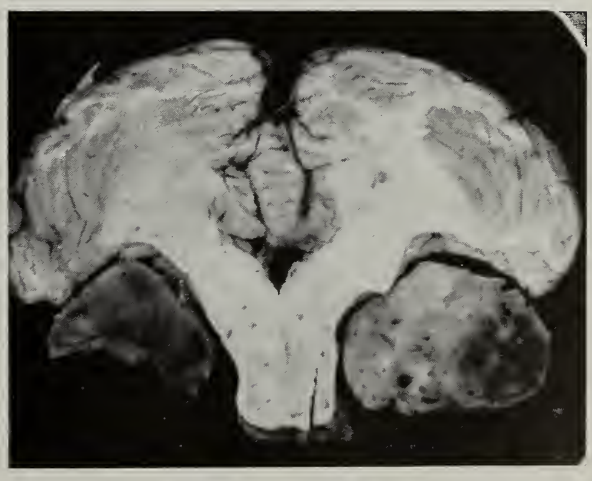


FIG. 4. Marked deformity of the cerebellar hemispheres and pons as a result of compression by the tumors.
H & E x 160

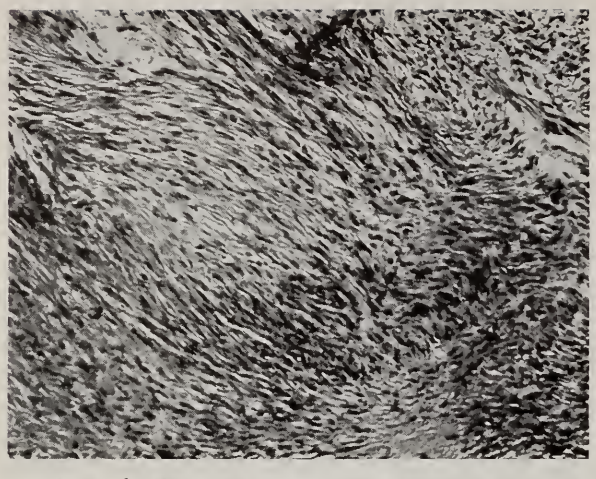


FIG. 5 Area of the tumor showing interlacing cellular arrangement and regimentation Antoni Type A.
H & E x 160

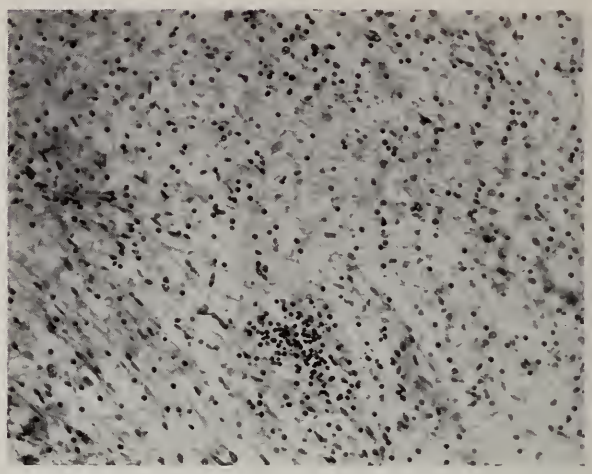


FIG. 6. Another area of the tumor with loose reticular pattern of Antoni B type.
H & E x 160

there was a smooth-surfaced, deep, nest-like excavation from which the tumors could be extracted with some difficulty. The pons was likewise markedly deformed, flattened, elongated, and deviated to the right. The tumor could be seen extending into both internal auditory meati, where infundibuliform pressure excavations of the petrous bone were clearly evident.

Both temporal lobes had herniated through the incissura cerebelli and there was marked coning of the cerebellar tonsils which had also herniated through the foramen magnum.

On the cut surface, the brain showed marked dilatation of the lateral and third ventricles which, doubtless, was the result of the compression by the tumors on the Sylvian acqueduct, the latter structure being greatly diminished in caliber and sharply angulated to the right in its trajectory (Fig. 3 and 4).

The cerebrospinal fluid was clear and colorless and the plexus choroideus showed no remarkable features.

A fine granular ependymitis was visible in the lateral and third ventricles.

When the tumors were sectioned, one could note, in areas, a definite whorled pattern. The texture of the tumors varied from firm to almost pulp-like in areas where hemorrhage was evident.

Microscopically, the tumors were typical neurinomas composed of the two types of Antoni architecture, A and B, with areas of regimentation in which all cell nuclei were basophilic and cigar-like in shape, occurred parallel to each other and at the same level (Fig. 5), while in others there was a reticular pattern in which cell nuclei were disarranged (Fig. 6). In places, one could encounter a number of large, dark, nucleated cells of the type mentioned by several authors.

The hemorrhages noted grossly were present, for the most part, in the vicinity of moderate-sized vessels. Some of these hemorrhages were quite recent, while others were of older occurrence and were depicted by brownish deposits of hemosiderin.



FIG. 7. Old and recent hemorrhages within the tumor which occupy a perivascular location.
H & E x 44

DISCUSSION

Schwannoma, neurilemmoma, neurinoma, neuroma, neurofibroma is an almost invariably benign, notoriously slow-growing tumor of the peripheral nerves, the spinal roots, and the cranial nerves. Though this tumor is frequently solitary, on occasions, multiple tumors may be encountered in different nerves, particularly in association with Von Recklinghausen's disease. These tumors may vary considerably in size from a minute swelling to enormous masses which may extend along the course of a nerve.

Intracranial Schwannomas form about 8% of all primary tumors of this region. They are more commonly encountered in females (Cushing, 1917, Gonzalez Revilla, 1947, Edwards, 1951, and Dorothy Russell, the latter author reporting an incidence of 76% in females).

When neurilemmomas are located in the cranium, the vast majority of cases are seen growing from the eighth nerve.

Although not the only type of tumors to grow in such an area, the frequency with which the tumors are en-

countered at the angle topographically located on the lateral aspect of the brain stem at the junction of the pons and medulla limited above by the biventral cerebellar lobule, has won for them the frequently applied name of cerebellopontine angle tumors.

The eighth nerve neurilemmomas arise, for the most part, from the vestibular portion of the nerve, though they may occasionally arise from the cochlear division according to Rockowitz, 1952.

As mentioned by Cushing and several other authors, and for reasons until now unknown, there seems to be a definite predilection for these tumors to occur on the left side.

Gardner and Frazier, who studied a family tree of 200 members, found among them 37 cases of neurilemmoma which leads him to believe that it may be transmitted as a true Mendelian factor. Gonzalez Revilla, in his review of the Johns Hopkins cases till 1947, makes mention of two sisters with a history of acoustic neurinoma where, on the maternal side of the family, he found 35 affected members which could be traced as far back as the 18th century, and Giudetti studied a family in which members of three generations were affected.

Neurilemmomas are more commonly encountered as single unilateral tumors; however, several cases of bilateral tumors have been cited, the great majority of these associated with generalized neurofibromatosis.

Our case we believe to be of particular interest, since none of the associated symptoms of Von Recklinghausen's disease were evident as in the cases of Mark and Sweet, or of B. W. Rovine and Mulford, although in the latter there were numerous associated meningiomas.

On gross inspection, the tumors are generally firm, circumscribed, and encapsulated, varying in color from a milky-white to semi-translucent. As in our case, areas of hemorrhage may be encountered in them and even more rarely, as in the case of Rohmer and Wackenheim, one may find associated areas of calcification.

Although these tumors may occur at any age, they are usually seen during the third and fourth decades, and are infrequently seen in children, MckCraig. Cushing found the majority at an average age of 44.4 years, Gonzalez Revilla at 38.1 years, Henschen at 44.0 years, Thorsrud, 40 years.

Another characteristic of neurinomas is their tendency to recur, this tendency apparently being related to the histologic type. Figures range, in published cases, from rare recurrence to those in which the latter occurred in over 50% of cases.

The etiology of these tumors still remains unsettled, some authors suggesting that the tumor arises from glial tissue, while others rather think that their most likely origin is from the sheath of Schwann, but an origin from the sheath of Schwann is accepted by the majority of authorities.

The typical histologic picture consists of cells with indistinct borders and elongated nuclei which are arranged in picket-fence fashion and form whorls.

Reference is often made to the two Antoni types, A and B, the first exemplified by a polar interlacing cellular arrangement with characteristic regimentation of nuclei, while the type B consists of a loose reticular tissue in an apolar arrangement. Though this is interesting, it has frequently been mentioned that, as occurred in our case, the two types may be found in the same tumor.

Although, as mentioned before, the tumors are generally milky-white, one often sees those which have a loose reticular tissue in which fat-laden foam cells have accumulated, resulting in shades which vary from pink to pale yellow or orange.

The vessels in the tumor often reveal thickening and hyalinization, and evidence of interference with the blood supply is not infrequently seen, exemplified by areas of hemorrhage, tumor degeneration, hemosiderin deposition, or cyst formation.

As in our case, acoustic neuromas of the eighth nerve frequently dilate the internal auditory meatus, this being an important feature employed by the radiologists for their diagnosis.

The symptoms most frequently encountered are deafness and tinnitus and only later do other symptoms appear.

When the tumor extends in a cephalad direction, the patient may complain of anesthesia or neuralgia referable to the area innervated by the fifth nerve, or there may be loss of corneal reflex or, if the tumor is of larger proportions, malfunction of the muscles of mastication on the affected side may occur.

When the tumor grows in a central fashion, symptoms of cerebellar disturbance may appear.

When, however, the tumor grows caudally, symptoms referable to the ninth and tenth cranial nerves will often appear, associated with diplopia. Finally, there may appear the so-called cerebellar crises and respiratory difficulties.

Pyramidal signs appear late and are not common unless there is indication of pressure in the brain stem.

In an attempt to evaluate the rather remarkable dilatation of the sella turcica present in our case, we have theorized the following:

With the growth of the tumor, the brain stem slowly and progressively became bilaterally compressed; this resulted in distortion and diminution of the caliber of the Sylvian acqueduct which, in turn, resulted in hydrocephalus that distended the floor of the third ventricle. The enormous dilatation of the sella turcica without erosion of the clinoid processes, therefore, is clear evidence that the increased hydrostatic pressure within the downwards herniating third ventricle occurred already in early life when the bones were still growing and the sutures were not closed.

ACKNOWLEDGMENT

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Erythema Infectiosum

Rowland B. French, M.D.

Erythema Infectiosum, or Fifth's disease, is a contagious disease, probably of viral origin, and appears sporadically in outbreaks. It occurs particularly in children ages four to twelve with an incubation period of six to fourteen days and is largely asymptomatic. It is characterized by a fairly brilliant red rash over the cheeks, spreading gradually over the extremities over a period of several days. The most outstanding feature is it's reticular pattern, suggestive of a rash following an intense, continued, hot application. The purpose of this report is to suggest that the disease would be quite common if, perhaps, there were an increasing awareness of it.[1,2]

Case Report

This is a report of eleven cases seen over a six month period. The first was a 7-year-old white boy who developed a confluent, retiform red rash over his cheeks, not involving the nose, and gradually spreading over the upper extremities and slightly on the chest over a several day period. The rash became more prominent on exercise. Examination revealed no fever; he was asymptomatic. There were no occipital nodes. Wbc, 9,500. Differential: 48% lymphocytes, 9% eosinophiles, 43% polymorphonuclears. Throat culture revealed no Betahemolytic streptococci, but Alphahemolytic streptococci and Coagulase positive staphylococci were found. An eleven-year-old brother had a similar rash one week prior to his onset, and a 4½-year-old brother developed a rash two days after his onset. Two weeks prior to his onset, a 7½-year-old girl who lives next door had a similar rash.

Seven other children developed a similar rash after his onset in the following sequence: two months later, a six-year-old boy living in the same town; three months later, three children, a boy four years old and two girls ages two-and-one-half and nine, of one family living about a block away, as well as another three-and-one-half year old boy living in the same town; five months later, a three-year-old boy from an adjoining town and six months later, a ten-year-old boy, also from an adjoining town.

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16 Water Street, Eastport, Maine

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Syphilis Serology Is Not Enough

CHARLES H. OKEY, PH.D.*

For several years, the Department of Health and Welfare has recommended to physicians that they prophylactically treat contacts with a history of recent exposure to cases of communicable syphilis regardless of the absence of lesions or a reactive serologic test for syphilis. However, this recommendation has apparently not been so widely proclaimed as it should have been, as evinced by the following report:

In mid-October of this year, an epidemiologic report was received from another New England State. It stated only that a seventeen-year-old white female (last name unknown), in a Maine community, had been recently exposed to a case of primary syphilis. The above contact had been picked up in front of a department store on the main street of the town. Her home address was unknown.

Follow-up of this case was handled by public health nurses, who located and brought the contact to diagnosis and treatment through a private physician within seven days. The diagnosis was primary syphilis.

When interviewed for her sexual contacts, the patient named three persons — one in another state (the original case); one who had recently moved to a foreign country (follow-up continuing); and one seventeen-year-old white male in an adjoining town.

The teenage contact was located by the public health nurses in the area and referred to a nearby hospital for examination. A serologic test for syphilis (STS) was negative at this time and the contact was discharged without prophylactic treatment.

In mid-November, this contact noticed a small penile lesion which grew steadily larger over the two-week period before medical examination was sought. In early December, a physician was consulted, who immediately drew a blood specimen for an STS and administered penicillin. The Department was called, and a darkfield microscope was taken to the doctor's office for an examination of the lesion. This examination proved ineffective, however, because of the recently administered penicillin. The quantitative STS result was later reported to be reactive to two dils, and the final diagnosis was syphilis in the primary stage.

This case of syphilis should not have developed. "If the patient is known to have been exposed to a lesion of syphilis, it is a fallacy to wait for the disease to develop to the clinical or reactive serologic stage, meanwhile allowing reinfection of treated patients and the infection of additional persons."[1] Since the incubation period of syphilis varies (10 to 90 days) from individual to individual, and since reagin tests for syphilis do not generally become reactive until after the appearance of the primary chancre, epidemiologic or prophylactic treatment is recommended for all contacts with a history of exposure to cases of communicable syphilis within the previous ninety (90) days.

A minimum total dosage for prophylaxis as recommended by the U. S. Public Health Service is 2.4 million units of either Procaine Penicillin G (with 2% Aluminum Monostearate) or Benzathine Penicillin G.[2]

The only method of absolute diagnosis of syphilis is the demonstration of the Treponema pallidum by means of the darkfield microscope. These microscopes are available to any physician in Maine for the examination of lesions; a telephone call to the District Health Officer or to the Department in Augusta will speed a microscope and a trained technician to the doctor's office.

While immediate treatment of diagnosed cases of early syphilis is highly desirable, penicillin or other broad range antibiotics should not be administered prior to a darkfield examination because less than 100,000 units of penicillin (given locally or systemically) will render such examination completely ineffective.

Other diagnostic and epidemiologic aids for syphilis

*Director, Diagnostic Laboratory.

[1] *Syphilis, Modern Diagnosis and Management,* Sidney Olansky, M.D., and Evan W. Thomas, M.D., Public Health Service Publication No. 743, July, 1961.

[2] *Notes on Modern Management of VD,* U.S. Department of Health, Education and Welfare, Public Health Service Publication No. 859, 1962.

Continued on Page 20

Report of Maine Medical Association Delegate on Actions of the House of Delegates of the American Medical Association at the Eighteenth Clinical Meeting, Miami Beach, Florida, Nov. 29-Dec. 2, 1964

At this 18th Clinical Session of the A.M.A., a shroud of sadness was apparent due to the untimely death of its President, Norman A. Welch, who died on September third.

President-Elect Donovan A. Ward took office after the death of Dr. Welch and carried on the business of this meeting with skill and efficiency.

Usually there are no elections at this Clinical Session but due to the aforementioned tragedy it was necessary to fill vacant offices. Dr. James Z. Appel of Lancaster, Pennsylvania was elected President-Elect of the Association, while Joseph B. Copeland of Austin, Texas filled Dr. Appel's place on the Board of Trustees.

Health Care for the Aging

Definitive action on the issue of health care for the aging came with the House of Delegate's strong endorsement of Dr. Ward's Monday address, in which he declared that, "We have no choice except to stand firm in our efforts to prevent the standards of health care in this country from being undermined by a radical departure from the unique American way which has accomplished so much for mankind."

Reaffirming the Association's opposition to the King-Anderson type of legislation, Dr. Ward said:

"If we have been right in the past — and that is our unshakeable belief — then we are right today. And, we shall be right tomorrow."

Calling for renewed intensive effort to prevent the passage of such legislation, he pointed out that "we do not, by profession, compromise in matters of life and death. Nor can we compromise with honor and duty."

Dr. Ward, expressing pride in the medical profession, concluded his address with these statements:

"I pray that we all gain strength for renewed effort by the simple reflection that what we are doing is worthwhile — that if the effort is great, the results of not making the effort would be unthinkable — and, finally, what we are doing is vastly more important than ourselves.

"No more can be asked of us as citizens. No less should be offered by us in guarding our heritage of freedom."

To implement the ideas in Dr. Ward's address, the House gave unequivocal approval of a Board of Trustees' suggestion that an expanded educational program be conducted in the next few months. In asking for this approval, the Board pointed out that "a variety of techniques and media must be utilized if the public, the Congress and special audiences are to be reached effectively."

Teletype Communications System

In discussing this system the following facts were presented.

a. A total initial cost of installing the equipment at A.M.A. Headquarters and at the headquarters of each participating State Society estimated at $1500 will be paid by A.M.A.
b. Total annual rental cost for equipment installed at Headquarters of A.M.A. and each of forty-nine State Headquarters estimated at $20,000 will be paid by A.M.A.
c. Cost of transmitting messages will be paid by the organization originating the messages. A nine hundred word message from Brunswick, Maine to Chicago would cost $3.70.
d. No extra office help should be required as anyone who can operate a typewriter can operate this machine.
e. This system will enable any state society to communicate with A.M.A. or with any other State Society Subscriber in U. S. or Canada without involving A.M.A. Headquarters.
f. No increase in A.M.A. dues is proposed at this time.

Considerable discussion among the Delegates as to the need for this service and its cost was noted and it was favorably voted by about 2 to 1 with your Delegate dissenting. However, it was made optional with each state whether they would install the system or not.

Human Reproduction

Updating its policies on population control, "to conform to changes in society and medicine" and to "take a more positive position on this very important medical-socio-economic problem," the House adopted the following four-point statement:

1. An intelligent recognition of the problems that relate to human reproduction, including the need for population control, is more than a matter of responsible parenthood; it is a matter of responsible medical practice.

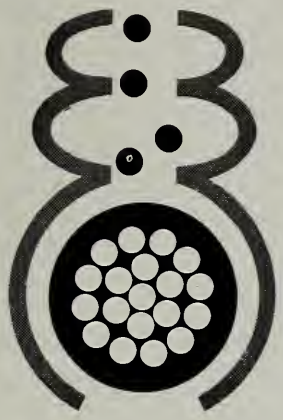
2. The medical profession should accept a major responsibility in matters related to human reproduction as they affect the total population and the individual family.

3. In discharging this responsibility, physicians must be prepared to provide counsel and guidance when the needs of their patients require it or refer the patients to appropriate persons.

4. The A.M.A. shall take the responsibility for disseminating information to physicians on all phases of human reproduction, including sexual behavior, by whatever means are appropriate.

In taking the action, the House also recommended that the A.M.A. cooperate with the appropriate voluntary organizations in the field of human reproduction which have adequate medical direction.

Continued on Page 20



LOMOTIL®

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride . . . . . 2.5 mg.
(Warning: May be habit forming)
atropine sulfate . . . . . 0.025 mg.

- lowers motility promptly
- relieves spasm promptly
- stops diarrhea promptly

Lomotil fulfills the first order of treatment in most patients with diarrhea — prompt symptomatic control.

Pending discovery of the cause, early cessation of diarrhea is almost always urgently indicated. Prompt symptomatic control averts distress, dehydration and, frequently, severe exhaustion.

Both experimental and clinical evidence indicates that Lomotil exerts such control efficiently, safely and with maximal promptness.

dosage:

The recommended initial *adult* dosage is two tablets (2.5 mg. each) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is controlled. Maintenance dosage may be as low as two tablets daily. *Children's* daily dosage (in divided doses) varies from 3 mg. for a child of 3 to 6 months, to 10 mg. for one 8 to 12 years of age.

cautions and side effects:

Lomotil is an exempt narcotic; its abuse liability is low and comparable to that of codeine. Recommended dosages should not be exceeded. Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdosage.

SEARLE

Research in the Service of Medicine

Maine Heart Association Notes

Rheumatic Recurrences In Patients Not Receiving Continuous Prophylaxis

"Rheumatic fever recurs less frequently as the rheumatic host ages, and recurrent attacks are observed relatively rarely in the latter part of life. . . .

"The results of this study have implications concerning the duration for which continuous prophylaxis should be maintained in the rheumatic host. Although rheumatic "hypersensitivity" to the Streptococcus appears to decline with age, as does the frequency of streptococcal infections, the adult rheumatic patient still can react, long after his previous rheumatic attack to a strong, streptococcal antigenic stimulus, with a recurrence rate several times that of the general population. In the presence of rheumatic heart disease this "hyperreactivity" is greater. Discontinuing antistreptococcal prophylaxis in the adult rheumatic cardiac still constitutes at least a potential serious risk, and one that should be taken only with full knowledge of the epidemiology of streptococcal disease in the patient's environment."

Reference: Johnson, E., Stollerman, G., and Grossman, B. JAMA, Volume 190, pages 407-413, 1964.

Submitted by Jacob B. Dana, M.D.

Low Cost Penicillin For Rheumatic Fever Prophylaxis

The Maine Heart Association, in conjunction with the Maine Pharmaceutical Association and the Maine Drug Wholesalers, and with the approval of the Maine Medical Association and the Maine Department of Health and Welfare, has initiated a program for making available to patients, who have had rheumatic fever, penicillin at low cost for rheumatic fever prophylaxis.

Any patient, private or clinic, with a diagnosis of rheumatic fever or rheumatic heart disease, past or present, is eligible. Diagnosis, in so far as possible, should be based on the modified Jones criteria.

For complete details on the program write the Maine Heart Association, 116 State Street, Augusta, Maine.

News, Notes and Announcements

Veterans Administration Center, Togus, Maine

To the Members of the Maine Medical Association:

On December 8, 1964 the Veterans Administration closed its outpatient clinic at 171 Middle Street, Portland, Maine. Veterans formerly receiving treatment for their service-connected disabilities at that clinic have been advised that treatment may be obtained from a doctor of their choice in the city or town in which they reside and will be authorized by the Veterans Administration Center, Togus, Maine upon the receipt of an application for the same.

The Veterans Administration earnestly seeks the cooperation of the medical profession in caring for its eligible veteran-patients. All physicians who have been duly licensed to practice medicine or surgery in the State of Maine and are citizens of the United States may participate in the Veterans Administration medical program. Physicians not holding an appointment with the Veterans Administration are cordially invited to apply for such by writing to the Chief, Outpatient Service, Veterans Administration Center, Togus, Maine requesting their appointment.

Recently the Veterans Administration negotiated with the Maine Medical Association for schedule of fees. With the approval of the Veterans Administration Central Office, Washington, D.C., the new fee schedule will be effective July 1, 1966. It replaces in its entirety the 1958 schedule and raises physicians' charges to the level now made by physicians to the general public.

LORRIMER M. SCHMIDT, M.D.
Chief, Outpatient Service
Veterans Administration Center
Togus, Maine

American Cancer Society, Maine Division, Inc.

The American Cancer Society, Maine Division, Inc., Federal Street, Brunswick, Maine, will be glad to receive applications for stipends to cover research projects in cancer from properly qualified individuals, or institutions.

Contact the Maine Cancer Society office in Brunswick for proper forms which will detail the scope of work planned and outlines for procedure.

All applications, when properly submitted will be forwarded to the Scientific Committee of the American Cancer Society, for evaluation relative to scientific and research value, and recommendation as to possible grant.

Final decision will rest with the Program Development Committee of the Maine Cancer Society.

Second Clinic for the Mentally Retarded Pre-School Child

A new clinic for the mentally retarded pre-school child will begin on January 11, 1965 at 9 a.m. at the Central Maine General Hospital in Lewiston. The clinic is sponsored by the Division of Maternal and Child Health of the Department. Dr. Russell Morissette, Lewiston will serve as clinic director. Assisting Dr. Morissette will be staff from the Division of Maternal and Child Health as follows: Mrs. Roberta Hanson, Speech Consultant, Miss Dorothy Woodcock, Nutrition Consultant and Mrs. Dorothy Morissette, Medical Social Consultant.

The clinic will operate on a monthly basis – the second Monday of each month at 9 o'clock. It is the second of its

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PSYCHIATRY FOR THE MEDICAL PRACTITIONER
New Orleans, Louisiana
March 4-6, 1965

Sponsored by the DIVISION OF NEUROLOGY AND PSYCHIATRY OF TOURO INFIRMARY under a National Institute of Mental Health Grant.

GUEST LECTURERS INCLUDE:

Jack Ewalt, M.D., Prof. of Psychiatry, Harvard Med. School, Past Pres. of American Psychiatric Association, Boston, Mass.

John Lambert, M.D., Medical Director, Four Winds Hospital, Katonah, N. Y.

Zigmond Lebensohn, M.D., Chief, Dept. of Psychiatry, Sibley Memorial Hospital, Washington, D. C.

William Sheeley, M.D., Director of Psychiatry & Medical Practice Project of the A.P.A., Washington, D. C.

Philip Solomon, M.D., Chairman, A.P.A. Committee on Medical Practice, Boston, Mass.

Course will be given at Jung Hotel, 1500 Canal Street, New Orleans, La. Hotel reservations to be made directly with the Jung or hotel of your choice. Registrants who would like to enjoy Mardi Gras (March 2) are urged to make hotel reservations *immediately*.

Guest speaker for the luncheon on March 4 will be George Burch, M.D., Henderson Professor and Chairman, Dept. of Medicine, Tulane Medical School. Subject: "Emotions and Cardiovascular Disease." Cost of luncheon included in registration fee. At the end of Friday's session, there will be a dutch treat two-hour cocktail party with George Lewis and his band from Preservation Hall entertaining.

AMONG TOPICS TO BE DISCUSSED:

"Detection of Incipient Psychiatric Disorders During a General Medical Examination"
"Medical Practitioners and Supportive Handling of Schizophrenia"
"Adolescents — Disturbed and Disturbing"
"The Physician and His Reaction to the 'Crock' "
"Newer Thoughts About the Therapy of Alcoholism"
"Medical Conditions with Psychiatric Manifestations"
"Recognition and Treatment of Depressive Reactions by Medical Practitioners"
"Treatment of Emotional States by the Medical Practitioner"

Gene L. Usdin, M.D., Chief
Division of Neurology & Psychiatry
Touro Infirmary
3516 Prytania Street
New Orleans, La. 70115

Enclosed is my registration fee of $20 for the "Psychiatry for the Medical Practitioner" course to be given March 4-6, 1965, at the Jung Hotel. (Checks should be made payable to Touro Infirmary.)

NAME .....

ADDRESS .....

.....

type to be established in Maine – the first clinic for the mentally retarded pre-school child having been started at Thayer Hospital, Waterville, in 1957.

All patients will be handled on a referral basis through the above division of service in the Department.

Correction

Kennebec County Medical Society Notes — November, 1964 issue, p 216, para. 4, line 1 should have read: "Alfred Hurwitz, M.D. was introduced by Bruce Trembly, M.D."

OFFICE MANAGEMENT OF THE EMOTIONALLY DISTURBED PATIENT — *Continued from Page 9*

Wittkower, E. D., and Cleghorn, R. A., (eds.), *Recent Developments in Psychosomatic Medicine,* Lippincott, Phila., 1954.

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Maine Medical Center, Portland, Maine

DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 15*

offered to physicians by the Department include serologic tests (both treponemal and non-treponemal); diagnosis consultation via telephone (City or District Health Officers); current professional literature; and patient interviews and contact follow-up.

The cases illustrated above are not atypical. The average patient with syphilis treated by private physicians in the State of Maine names more than three (arithmetic mean) sexual contacts during the critical period of the infection, and numerous investigations have shown that 35% of all contacts are likewise infected with syphilis. This percentage rises dramatically, however, if prophylaxis is denied asymptomatic contacts.

Syphilis morbidity reporting is increasing in Maine; morbidity reports received so far this year have increased by more than 40% when compared with the receipts for the calendar year 1963. This increase may represent more complete reporting on the part of physicians, or it may reflect an actual increase in new cases. In either event, vigilance is indicated as well as the utilization of all of the diagnostic and epidemiologic aids available if the five-century-old battle against *Morbus Gallicus* is to be won.

REPORT OF DELEGATE — *Continued from Page 16*

MISCELLANEOUS ACTIONS

In considering a wide variety of annual reports, special and supplementary reports and resolutions, the House also:

Amended the Bylaws to permit the presidential inauguration to take place at a time other than Tuesday evening and approved a suggestion that the inaugural ceremony at the 1965 Annual Convention to be held on Sunday, June 20;

Urged strong support of the Woman's Auxiliary and asked the state and county medical societies to give serious consideration to the idea of joint husband-wife membership;

Agreed that a section on Space Medicine should not be created at this time;

Emphasized its continuing awareness of the demand for action on satisfying the need for increasing numbers of family physicians;

Approved a Board recommendation that the 1967 Clinical Convention be held in Houston Texas;

Reaffirmed its approval and support of the National Council for Accreditation of Nursing Homes and

Instructed the Board to reevaluate the mission of the Commission on Medical Practice and take appropriate action.

The American Medical Association Education and Research Foundation reported to the House that one out of every six medical students, interns and residents in the U. S. is now receiving financial assistance from the Foundation's loan fund. The AMA-ERF also announced that Merck Sharp & Dohme pharmaceutical company has made its fourth $100,000 contribution to the loan fund and has pledged an additional $100,000 in 1966.

This is my final report as your delegate to A.M.A. This has been a rewarding experience and it is with a certain feeling of reluctance that I pass my baton over to my successor.

I am sure that the many friendships that he will make and the contributions that he will give to Organized Medicine will be one of the greatest experiences of his career.

ASA C. ADAMS, M.D.
Delegate to A.M.A.

The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, February, 1965 | No. 2

Cystic Lesions of the Parotid Region

Loring W. Pratt, M.D.*

Swellings and masses in the region of the head and neck often have an ominous significance. For this reason their differential diagnosis is of great importance to the patient, and of considerable help to the physician.

Cystic lesions of the parotid are in general benign and are of less serious consequence than solid masses found in the same area. Their differentiation, however, has considerable importance in the management of problems in this region, and their nature must be understood in order to make a satisfactory diagnosis.

The first group of swellings to consider would be congenital lesions. These result from faulty fusion of branchial arches one and two, giving rise to branchial cleft cysts of considerable variety. Because of the variations possible from their embryologic origin they may range from a preauricular dimple to a tubular connection between the preauricular region and the pharynx. Obliteration of either extremity or of both extremities of this tubular structure permits cystic dilatation of the vestigial sac and this in turn, when filled with fluid, is called a branchial cleft cyst. These are relatively common, and it is of importance that their embryology be understood in order that superficial excision of only a portion will not be attempted, but that complete extirpation of the entire tract, without injury to the nearby facial nerve, will be carried out.

True cystic lesions of the glandular tissue of the parotid are rarely found to be congenital lesions. The literature is almost devoid of specific references to congenital cystic lesions of the gland proper.

The second group comprise acquired cystic lesions of the parotid gland. These in general are multiple small cysts of the terminal ducts rather than large loculated cysts of solitary nature. There is one report of a solitary cyst which contained 17 cc. of fluid, but this is unusual. There are many instances of multiple small cysts which appear throughout the parotid tissue and these vary from tiny dilatations of the terminal ductal systems to rather large, multiple cystic areas which are properly termed sialiectasis. These are usually the result of inflammatory disease and result from ductal obstruction due to inflammatory changes, with subsequent development of cystic dilatations in the peripheral parts of the ducts.

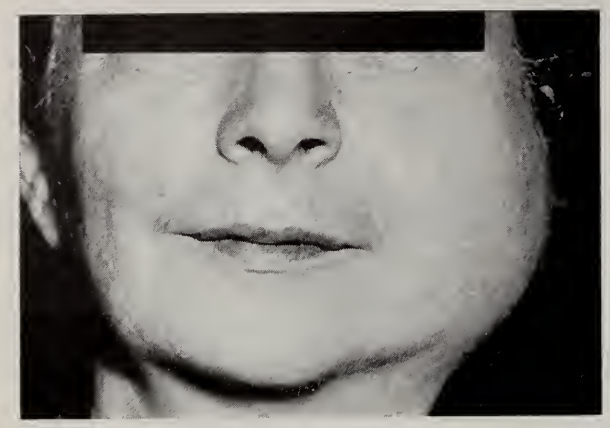
Inflammatory lesions which contribute to ductal obstruction are primary, and infections ascending Stenson's duct are secondary and are the proximate cause of the cystic disease. Primary infections are, for example, stomatitis, dental extraction, dental infection, peritonsillar abscess, retropharyngeal abscess, and infection of an adjacent lymph node. These, in turn, give rise to the secondary infection of the ducts with ductal obstruction.

Calculi may be the primary cause of the ductal obstruction with secondary ascending infection of the duct system. Although the etiology of the calculus may be uncertain, its effect is well established in producing ascending infection, with consequent partial obstruction and generalized or localized cystic dilatation of the gland.

Sjogren's Syndrome is one of the classical inflammatory diseases of the parotid and other salivary glands, sometimes called Uveoparotid fever or Sicca Syndrome. It is manifest by recurrent, intermittent swellings of the salivary glands, which fluctuate in size and consistency and are often found to be somewhat tender during the periods of engorgement. The secretion is viscid and does not readily flow from the gland. Similar swellings, in association with swellings of the lachrymal apparatus, are termed Mikulicz's Disease. The current thought on Sjogren's Syndrome is that it is a systemic disease, possibly related to the collagen disorders. Microscopically the lesion is one of small cell

*Thayer Hospital, Waterville, Maine

FIG. 1



a. Front view of parotid swelling caused by mandibular dentigerous cyst.



b. Lateral view of the same patient.

infiltration of the gland with ductal obstruction which in turn produces small saccular dilatations throughout the substance of the gland. Secretory sialography shows that delayed emptying of these cystic areas is a prominent feature.

Epidemic parotitis may, but rarely does, produce sufficient inflammatory change in the ductal system of the gland to produce subsequent cystic changes within the gland.

Trauma to the parotid and its adjacent tissues is an unusual reason for the development of cystic areas within it. Although cicatricial stenosis of the ducts from penetrating wounds does occur; except for war wounds, this is a relatively uncommon problem in medical practice. There are, however, many cases on record in which shell fragments and shrapnel, both intraglandular and intraductal, have contributed to the development of cystic lesions in this area. Complete obstruction of the duct produces glandular atrophy while partial obstruction gives rise to cystic changes.

A rare swelling in the parotid is the gasseous cyst of Stenson's duct, found in glassblowers and musicians who specialize in wind instruments. It is reported that these swellings may attain the size of a hen's egg.

Superficial cystic lesions in the form of epidermoid

FIG. 2



a. Lateral view of sialogram showing multiple cysts of parotid gland.



b. A.P. view of same sialogram.

cysts are often found in this region. They are less frequent here than in many areas of the skin, but must always be considered in the differential diagnosis.

The differential diagnosis of these lesions is important and it must be made with care. In the first place, there are a number of apparent cystic lesions which are not really cystic. For example, lipomas and enlarged lymph nodes in the region or even within the substance of the parotid may present a cystic appearance and feel. Mixed tumors of the parotid are at times cystic in some areas, but these are primarily neoplastic rather than cystic

lesions. The malignant tumor which undergoes necrotic changes within its mass may present a cystic sensation to the examining finger and yet not be a truly cystic lesion of the parotid. Cystic lesions of the adjacent dental structures may at times present the appearance of a parotid tumor, thus completely confusing the picture.

The illustration is that of a 52-year-old female, who had noted the gradual appearance of a mass in the parotid region over a period of two years. (Fig. 1, a, b) It was apparently firm and attached to the deeper tissues. In its terminal phase it became slightly tender. There was no sign of facial paralysis. The lesion was identified, at last, as a dentigerous cyst which had ruptured medial to the parotid gland and thus produced a deformity suggestive of a malignancy.

Secretory sialography is of great help in delineating the ductal system of the salivary apparatus and may be accomplished with relatively little difficulty. The technique of parotid sialography is simple. It consists of inserting a blunt 20 guage needle or a piece of #90 Polyethelene tubing into the anesthetized orifice of Stenson's duct and then injecting radiopaque material slowly, until there is appreciable dilatation of the gland and a sense of fullness or mild discomfort on the part of the patient. X-rays are taken in the lateral and anteroposterior planes immediately after filling. (Fig. 2, a, b) After injection and exposure of satisfactory x-ray films, the patient is given lemon to empty the gland. A film is taken in 15 minutes to study the post-evacuation status of the glandular ductal system. Incomplete emptying of the gland may well be as important as improper filling, as in Sjogren's Syndrome.

Conclusion

1. Many etiologic backgrounds account for cystic lesions of the parotid gland region.
2. Most of these lesions are acquired and arise secondary to chronic inflammatory changes in the parotid duct system.
3. Neighboring structures, such as the dental apparatus, must not be neglected in the search for a primary source of such diseases.
4. Sialography is of help in establishing the nature of these lesions.
5. These lesions are rarely malignant.

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Controls Must be Informed — Like Patients

It is worth noting that the law. . . (now) requires the investigator of new drugs to inform "any persons used as controls" that "**drugs** are being used for investigational purposes." The term "drugs" is confusing because it could refer to the new drug itself or to placebos or standard drugs given to the controls. Disclosure of full information to the controls may be highly detrimental to the drug study. Such disclosure introduces a highly undesirable psychologic element, inconsistent with the statutory requirement for adequate and well controlled studies for proving the effectiveness of a new drug. The conclusion is inescapable. The federal regulations and, if need be, the law, should be modified, so as not to interfere with medical practice and the orderly investigation of new drugs. — Francis Boyer, Chairman of the Board, Smith Kline and French Laboratories, in **New England Journal of Medicine,** 270: 15, (April 9,) 1964.

First Aid and Prophylaxis for Insect Stings

Samson Fisher, M.D.*

An estimated twenty[1] lives are lost annually in the United States due to fatal anaphylactic reactions from insect stings. Probably many other such deaths are mistakenly attributed to other causes. It would be difficult to estimate the frequency with which severe, life-threatening, but nonfatal reactions occur. In rural areas they are undoubtedly a frequent occurrence. Since repeat stings may elicit increasingly severe reactions, and since multiple stings rather than a single sting may occur, a life-threatening situation may predictably arise in susceptible individuals. It therefore becomes urgently necessary for these people, their families and physicians, to be familiar with first aid and prophylactic measures for insect stings.

Stinging insects are all in the class of Hymenoptera; and by their popular names include bees, wasps, hornets, and yellow jackets. Ants are also in this class, and are capable of inflicting a sting; but apparently their venom is not especially potent.

An allergic reaction to a sting may be local, with pain, itch, redness, and swelling. Beyond the discomfort experienced, this type of reaction is of little immediate consequence. Generalized, systemic, or constitutional reactions are of two types. Rarely seen is the delayed reaction with the typical manifestations of serum sickness. The second type, an immediate anaphylactic reaction, is a more common occurrence, and may demand quick, heroic treatment. This reaction may vary from the very mild, manifested by a few hives, to a rapid, violent, fatal outcome.

Life-threatening, near-fatal reactions are of three types. These are: 1. Massive edema involving the upper airways, with possible laryngeal obstruction. 2. Asthma may occur, with bronchial spasm, bronchial edema, and the outpouring of bloody, frothy mucus. 3. Anaphylactic shock may occur with circulatory collapse. Any combination of effects may occur simultaneously. It is important to be alert to the fact that in the presence of an easily recognized, dramatic reaction, such as urticaria or asthma, a more sinister and dangerous process may be in operation, such as edema of the larynx or shock. In fact, it may not be quickly evident that the patient is suffering from shock rather than an attack of simple syncope.

The treatment for anaphylactic shock, away from a hospital, may be limited to resting in a head down position, with protection to preserve body warmth, and efforts directed at maintaining an adequate air way. If facilities are available, intensive routine procedures for contending with shock may be indicated, including maintaining blood volume and an effective blood pressure; the use of oxygen; resuscitative and general supportive measures. Treatment problems arise when asthma and edema occur in the presence of shock. Medications usually administered for these conditions, such as epinephrine, aminophylline, and antihistamines may be specifically contraindicated in the presence of shock.

To contend with the classical allergic reaction, the patient or a member of the family can be instructed to carry out the following first aid procedures:

1. Epinephrine by injection is the drug par excellence. It can be injected in doses of 0.3 cc., and safely repeated every fifteen minutes, if necessary, for three or four doses. A dose can be injected directly into the site of the sting, so that the resulting vasoconstriction will retard absorption of the venom. If a tourniquet has been applied to an arm (v.i.), then the injection is preferably given in the opposite arm, or proximal to the tourniquet. Avoid injecting into an area of edema or a layer of fat; since rapid absorption is desired. Absorption will be hastened by massaging the site of injection. Epinephrine, or its analogues, as a spray, may be used. The dose is two to four deep inlalations.
2. Honeybees alone of all the stinging insects leave the stinger and the venom sac in the skin. This should be removed by scraping the skin with a dull blade, or possibly a long fingernail. Grasping the sac is to be avoided; since this forces more venom into the skin.
3. Isuprel® linguets may be used effectively; but it is difficult for small children, or anyone in a state of panic, to use this medication without sucking or swallowing. Isuprel is not effective to combat shock.
4. If the sting is on an appendage, a tourniquet can be applied proximal to the sting site.
5. Ice, cold packs, or an ethyl chloride spray can be applied directly to the sting site, to retard absorption of venom.
6. Lie down and rest quietly; or if feasible, proceed to a hospital and await further developments there.
7. In the presence of laryngeal edema, or vomiting, heroic measures may be necessary to maintain an airway.
8. Antihistamines and corticosteroids are relatively slow to take effect, and are no substitute for epinephrine. They may be given to achieve or maintain a prolonged anti-anaphylactic effect, with the knowledge that there may be a delay of minutes to hours before response occurs to these drugs.

Once the acute episode is out of the way, the question

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arises regarding the desirability or necessity for taking preventative measures to guard against a recurrence. This is not an easy decision to make. Hyposensitization, although effective, is tedious and injection treatment is indefinitely prolonged. Efforts directed at avoiding exposure to insects can be effective; but entails a severe restriction in activities. Less demanding measures are at best haphazard.

When should maximum preventative measures be carried out? An arbitrary rule is to include patients who suffer one serious reaction; and to include patients who have had more than one reaction, however mild, but the last one being much more intense than the preceding reactions. One's judgment in categorizing patients will undoubtedly be influenced by an estimation of the further likelihood of exposure. An urban adult may go many years or a lifetime without suffering an insect sting. Gardeners, farmers, sportsmen, and children who play in fields, on the other hand, have innumerable opportunities for exposure to single insects or swarms.

The general experience has been that severe reactions are not likely to occur without one or more previous reactions of less intensity. Hence, it is probably safe to caution and keep under observation the individual who suffers nothing worse than mild hives. Such patients should be instructed in first aid, and maybe given a first aid kit.

Prophylaxis for insect stings divide into those measures taken to avoid encounters with stinging insects, and those procedures that may prevent reactions after a sting has occurred.

After a sting occurs, all the procedures advised to contend with a reaction, are equally effective to prevent a reaction. Steps one through six, previously listed as treatment, may be carried out. In addition, the patient should be given one or more first aid kits, to be kept in the glove compartment of the car, in the home, and as part of an all purpose first aid kit. Such a kit should include:

1 Isuprel tablets (these may also be carried in the wallet in a flat pill container).

2. A hypodermic syringe and an ampule of epinephrine (loaded syringes have been on the market off and on; but none could be located at present).

3. Antihistamine pills.

4. A tourniquet, large enough to go around the thigh.

5. Printed directions for carrying out treatment procedures numbers 1 through 6.

And finally, hyposensitization may be indicated. Hyposensitization may be resorted to when it is not strongly indicated, to resolve the problem of what to do with the patient who is not understanding or dependable, and therefore incapable of carrying out first aid. Venom vaccines for testing and treatment are available commercially. There is no easy way to evaluate the degree or duration of immunity produced by this type of vaccine; so it is necessary to follow an arbitrary, established treatment schedule.[2] Experimentally there are effective procedures for giving multiple doses of vaccine in a single day, annually; or for giving a single injection of an emulsion preparation annually. These are reported to give effective and prolonged levels of immunity, and go far toward simplifying the treatment. However, this type of rapid treatment is not yet generally available.

Summary

Fatalities from insect stings are rare, but probably more frequent than vital statistics indicate. However, frightening and life-threatening anaphylactic reactions occur; and recurrences of such reactions are a fearful prospect.

If insect sting reactions are inconsequential or mild, it is sufficient to acquaint the individual or his family with the usual first aid procedures for insect stings; and to reassure the family that a more disturbing reaction should occur as a warning, before a severe reaction occurs.[3]

If a reaction of questionable severity occurs, the subsequent course of action depends on: 1. The intensity of previous reactions. 2. The estimated ability of the individual to contend with a severe reaction. 3. The estimated likelihood of future exposure to stinging insects. If the evaluation suggests that a conservative course is indicated, instruct the patient in first aid, and help him to prepare first aid kits. If in doubt, do this plus giving venom vaccine on a long term schedule.

If one severe anaphylactic reaction has occurred, then maximum prophylactic measures are indicated, including instruction in first aid plus routine vaccine treatment.

It is advisable to give all patients, in whatever category, and their families, a printed form enumerating the essential features of first aid, such as the following:

First Aid For Insect Stings

1. Take an injection of Adrenalin®. Inject 0.2 or 0.3 cc. into the site of the sting, and the same amount elsewhere. (Put this second injection *above* the tourniquet if a tourniquet is applied, or preferably put it in another arm. Massage this last injection site gently to hasten absorption.)

2. If Adrenalin is not available, put a pill (Isuprel) under tongue or between cheek and gum. Let it absorb. Do not suck or swollow it. Repeat once. After that, if a favorable effect has occurred, and then appears to wear off, repeat as necessary.

3. Put a tourniquet *above* the site of the sting, if it is on an arm or a leg.

4. You can put an ice pack on the site of the sting.

5. Go to a hospital or doctor's office if handy, to await further developments. Otherwise be quiet and rest.

6. You can take Benadryl® and cortisone by mouth or injection. These are *slow* to take effect; but are good for the delayed effects of the sting.

7. Try to identify the insect. If this is not possible,

at least take careful note of the surroundings where the encounter occurred.

To Avoid Exposure To Stinging Insects
In The Future, Take The Following Precautions:

1. Avoid areas where insects are encountered. Play in a playground or on beaches, rather than fields and gardens.
2. Use insect exterminators in and around premises.
3. Have someone get rid of known hives and nests from home surroundings.
4. Drive car with windows closed, and get air through screened ventilators, to keep winged insects from interior of car.
5. Certain individuals may be well advised to wear a MEDIC-ALERT emblem.

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Who's Killing General Practice - Is General Practice Necessary?

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In the June 15, 1964 issue of *Medical Economics* someone using the pen-name of Conrad Newhouse, M.D. observes that specialists are killing off general practice "by poisoning the public mind and destroying first the image and finally the reality of general practice." Indubitably there is competition between specialists and generalists for the best image in the public mind; this will always be, and one can only hope that fair means are employed by the contestants. As for the destruction of "the reality of general practice" (at first glance this reads like something to have investigated by the homicide squad) this is another matter and one which raises several interesting points for discussion.

Is there a real need for general practitioners? The answer to this is a qualified "yes." There is a definite and urgent need for what can best be called family physicians — trained in a two-year postgraduate program such as the one at the University of Kansas Medical School. In this program the major emphasis is on medicine, pediatrics and psychiatry; there is minor emphasis on medical obstetrics and gynecology, medical ENT and acute trauma. Major surgery is purposely left out of such a program. It has been claimed on good authority that 80% of the ills of the American people could be suitably treated by one so trained.

There are some, like the head of a large west coast clinic, who see no need for general practitioners, claiming that young internists can perform their duties. As a board internist with straight medical intern and residency training, it is hard for me to visualize an internist doing many of the jobs which g.p.'s do routinely: delivering babies, lancing ears, treating fractures and other trauma — to name a few. Obliging the patient to see another specialist for problems such as these seems impractical and costly.

The administrator of a famous east coast hospital advocates psychiatrists as the ideal general practitioners. They alone, he claims, are aware of the patient's entire being and are in the best position to call on other specialists when necessary. The same arguments that apply to the internists are cogent here. Admittedly the psyche is affected whenever the soma is disturbed, but the short supply of psychiatrists available to treat the psychotics obviates any possibility of their taking on extra duties such as this — granting that it should ever prove desirable.

From the practical standpoint it is hard to visualize American medicine practiced without a sufficient number of practitioners trained to treat the common afflictions. To state this in another way, the family physician, trained in the two-year program described above, would be more "at home" than the internist when treating minor surgical, GYN and ENT problems just as he would be better able to treat medical problems than the surgical specialist.

Can the family physician training programs succeed? Unfortunately, there are few accredited programs of this type and they are only half-filled. The majority of medical school graduates, trained in large teaching centers by prestigious specialists, follow in their masters' footsteps and shun the unglamorous robes of the gen-

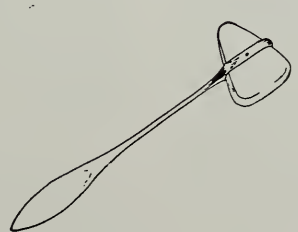
eralist. There are advantages in general practice which need more emphasis. The general practitioner sees a wide variety of problems, often requiring imagination and ingenuity in their disposition. He can escape the tyranny of the telephone by joining a colleague or two for night and weekend coverage. He is in a good position to make clinical discoveries of significance. Generalists have made excellent epidemiological surveys. Others have gained wide recognition as authorities in fields such as poliomyelitis or athletic injuries. He is able to adjust his practice to communities large or small. He is the most independent of practitioners. He is closer to his patients than most specialists and able to observe them in the family setting not only when ill, but frequently when at work or at play. These advantages may seem of less importance to the candidate who has gained the impression that the operating room is the profession's most lucrative arena. This consideration may steer some medical graduates into a rotating internship (with more surgical training) instead of into the family physician programs. One can assure these mercenary chaps (who might, in the end, have been happier with a banking career) that if present trends continue the financial rewards of general surgery will dwindle. At the moment surgical and medical residents are in a 1:1 ratio with a tiny number of family physician residents to add to the medical side. By conservative estimates this ratio in actual practice should be 1:4, surgical to medical.

What can be done? State legislation obliging a certain percentage of medical school graduates to enter family practice smacks of a totalitarianism foreign to the American way. The American Medical Association has established an ad hoc committee of the Council for Medical Education consisting of representatives of the Council for Medical Education, the American Academy of General Practice, the Association of Medical Colleges and the A.M.A. Section on General Practice. This committee is exploring ways to reinforce the shrinking numbers of general practitioners. A proposed recommendation is to have more medical student and intern training at the community hospital level rather than the large teaching center. After all, most Americans receive their medical care in community hospitals and, in these days of rapid communication and transportation, there is no reason why the level of care in these hospitals should not be on a par with the large teaching centers. This arrangement would benefit the trainee in several ways. He would become more familiar with the treatment of everyday medical problems and less absorbed by exotic maladies which he may rarely, if ever, encounter in a lifetime practice. He would be exposed to the socio-economic problems of medical practice which are assuming an increasing significance in the doctor's life and are often ignored in the university teaching centers. The trainee would also help the local staff by describing to them some of the never developments in medical knowledge; while his presence would inspire the local post-graduate teaching program, often carried on valiantly in the face of an apathetic staff.

Finally one wonders about the admission policies of the medical colleges. Are they overemphasizing intellectual prowess? Does one need an I.Q. in the genius range to practice good medicine? Is it conceivable that some of the poor medicine being practiced is by men of high intellectual calibre who are bored by the lack of intellectual stimulation in the day to day conduct of medical practice? Certainly the job of the admitting committees is not easy. The ideal candidate combines scientific curiosity and intelligence with a humanitarian desire to relieve suffering. Perhaps if the dedicated family physician is to be reborn there needs to be a shift in emphasis towards the last characteristic.

In summary, Dr. Newhouse has implied that specialists are killing off general practice. Speaking as a board internist I hope that family practitioners, trained along the lines of the University of Kansas two-year program, will survive. Hopefully the American Medical Colleges and the committees of the A.M.A. will find the way to preserve this worthy member of the medical community.

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Do Family Services Clients Have Rehabilitation Potential?

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Introduction

This is a report of a project which is a cooperative effort between the Division of Family Services and the two Rehabilitation Centers in Maine. The project which is ongoing began in September 1963. This is an interim report based on experience with 77 patients who had completed their evaluation by December 1, 1964.

The Division of Family Services within the Department of Health and Welfare is the agency responsible for administering public assistance programs — Old Age Assistance, Aid to the Disabled, Aid to the Blind, Aid to Dependent Children, and Medical Assistance for the Aged (Kerr-Mills). It has responsibility not only for the provision of financial services but also to provide services which will assist the individual client and family achieve their potential.

Patients referred by this Project to the Hyde Rehabilitation Center or the Thayer Hospital were not necessarily diagnostic problems. They were problems in adaptation to disease or handicap. Thus, the medical work-up was done not just to confirm diagnoses, but also to make a clear evaluation of the ability to adapt. In essence, we marshalled evidence to show the expected natural history of the patient's disease, his chances of adapting to the disease through medical treatment and also his own efforts, and an assessment of his reserves once he had compensated to his disease as well as possible. His capacity to return to some form of work depended on these reserves.

Methodology

Possible referrals to the project were screened from the entire State Aid to the Disabled caseload and the Aid to Dependent Children caseload in which deprivation of parental support was based on incapacity. The Family Services Medical Review Team, consisting of a physician and a medical social worker, retained responsibility to screen and select those likely persons who were considered Project referrals. Criteria were established to assist in the screening and selection. The team also assumed responsibility for determining which hospital facility was to be used for the evaluation. Based on the decisiveness of the background information, the patient was referred either for further diagnostic evaluation or for an intensive rehabilitation treatment program or both.

Formal Project referrals became the responsibility of a full time medical social worker who made a psychosocial summary, prepared the patient for the hospitalization, and acted as liaison between the agency, hospital, and other agencies which might have become involved, particularly the Division of Vocational Rehabilitation and the Maine Employment Security Commission.

On admission to the treatment center, the usual diagnostic work-up was completed not only for the purposes of confirmation of diagnosis but also to understand the natural history of disease and the present stage of the disease. Following this, the patient's adaptation to his disease was studied. If symptoms were more disabling than could be explained by abnormalities of structure or function, it was clear that the patient's adaptation was not appropriate and measures were taken to improve it. Finally the patient's reserves were studied. In patients that were disabled for any length of time, physical, emotional, and vocational disuse atrophy were frequently found.

In these studies, full use was made of medical and paramedical consultations. When the studies were completed, an evaluation conference was held with the patient to discuss his prognosis and future functional status. At the Thayer Hospital, medical consultants were members of the staff and therefore readily available to attend conferences. At Hyde Rehabilitation Center, the majority of consultations were arranged in the greater Portland area, some 40 miles distant, which precluded the consultants' participation in conferences. The patients' family physicians were often contacted by telephone and also received detailed medical reports. A detailed action program was developed based on the evaluation. Successful completion of this action program depended on follow-up done in cooperation with various agencies, particularly those in the patient's area of residence.

Observations

Disability for work is not synonymous with disease. It is well-known that patients react differently to disease. Some can continue in the face of severe handicaps while others are disabled by only minor handicaps. However, the decision of disability is often made solely on presence or absence of disease without equating its magnitude to patient adjustment factors.

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An excellent example of a discrepancy between presence of disease and presence of disability is a case of a 47-year-old man who was evaluated because of cardiac disease and digestive symptoms. In 1954, he had a myocardial infarction and was told that he had to give up work at that time. In 1955, he was operated and pericardial poudrage was carried out. Angina subsided to the point where he was able to obtain work as a toll collector. In January of 1963, he had an episode of gastrointestinal bleeding and stopped work at that time. On diagnostic work-up he was found to have coronary artery disease, active, following pericardial lysis and poudrage in 1955. He had antral gastritis with an active duodenal ulcer and a deformed bulb. In addition, he had cirrhosis of the liver and cervical osteoarthritis. At evaluation in September 1963, it was evident that none of his diseases would be helped by continued inactivity. It was recommended that he have active medical treatment and that he return to work. He returned to his job as toll collector and has been successfully employed ever since.

A case illustrating the converse of the above is a 29-year-old father of five children evaluated in February of 1964 for back pain. After full diagnostic work-up, no cause could be found for his low back pain except reflex muscle spasm. This was explained to the patient and his family by the doctor and the medical social worker over and over again in very simple terms. An intensive physical medicine program was carried out, but in spite of this he has never returned to employment.

The patients present an interesting breakdown as regards age and sex distribution. Of the 77 patient evaluations completed, 16 were female and 61 were male. The ages ranged from 23 to 61, the mean age being 42. Of these 77 patients, 25 were found to have no rehabilitation potential. Of the remaining 52, 20 were found not to have enough disability to be eligible for assistance, and their grants were closed. Of the remaining 32 found to have rehabilitation potential:

4 have returned to work

10 are in training or awaiting suitable job placement

4 are still under active medical treatment

14 are in planning stages or follow-up has broken down

An example of this last category is Mr. A., a 45-year-old widower, who was referred to the Project for a diagnostic evaluation. The psychosocial summary indicated this man to be of a minority race, and that despite severe cultural deprivation and family problems, he had a relatively stable work history. With these social factors in mind, it was recognized that numerous contacts would be necessary to encourage Mr. A. to move into some appropriate employment. The medical evaluation indicated a pulmonary problem of moderate nature which would not preclude the performing of many jobs for which he had competence. Rehabilitation potential seemed apparent. With the client's help, a cooperative plan of action was agreed upon which would enable him to obtain employment in a more urban area. Through lack of adequate or appropriate follow-up, an arranged employment interview was not kept and in the meantime, Mr. A. moved and left no forwarding address.

The humanitarian factor of getting even one patient back to productive living is self evident. In addition the monetary value of such a program is and will be of prime importance to tax payers. Such an example is Mr. B., age 32, who has been disabled and known to Family Services for over 10 years. Assistance to this family during the past fiscal year was approximately $3,000. This man has the potential for more than 30 years of productive employment. His rehabilitation will result in savings to the tax payers of Maine of more than $90,000.

Summary

This is a report of a project planned jointly by a State agency and two non-proprietary institutions.

The purpose of this project is to determine the potential of selected recipients and to provide the necessary rehabilitation treatment.

Our experience shows that disability and disease are not synonymous.

Present results of 77 patients whose evaluations have been completed are given.

This interim report shows that some Family Service applicants and recipients do have rehabilitation potential. It is hoped that this report will stimulate interest and cooperation of the practicing physicians, social workers, counselors, and any other tax payers interested in rehabilitation.

Dystonia Phenothiazorum

Richard T. Chamberlin, M.D. and Bruce Trembly, M.D.

Neuromuscular reactions to prochlorperazine (Compazine®) have been reported in the American literature intermittently since 1957,[1,2,3] but many physicians may not be aware of the significance of the peculiar group of symptoms that characterize the phenothiazine reaction. The syndrome is so characteristic that it can be readily recognized, and thereby hazardous and unnecessary studies and treatment measures can be avoided.

The following four cases have been observed by the authors at Thayer Hospital within the past year and serve to illustrate the syndrome of the phenothiazine reaction. They also imply its relative frequency in the general population. The purpose of this report is to again call attention to the syndrome as it may appear in the general setting of medical practice, emphasizing that these patients received phenothiazines in the course of treatment of common and "simple" symptoms and were not receiving the medication in the course of psychiatric treatment.

Case Reports

Case 1 (R.H. #41390): A 32-year-old male with a negative neurologic history was given Compazine because of a gastrointestinal upset. On January 15, 1964, he received 10 mg. by mouth four times. On the following day he received 10 mg. by mouth three times, the last dose being at six p.m. One to two hours later he developed a tight feeling in his neck and shoulder muscles, restlessness, severe apprehension, sweating, involuntary movements of his neck, twisting of his neck and trunk, and flexion of his arms and legs. His examination on admission that evening was normal except for involuntary movements which came on in waves and consisted mostly of extension of his neck and back with spasm of his sternocleidomastoid and platysma muscles on both sides. He was given Sodium Amytal,® 3¾ gr. intramuscularly twice during that evening, and by early the following morning he was asymptomatic. There have been no subsequent symptoms.

Case 2 (J.H. #41806). A 16-year-old school girl was given Prolixin® (fluphenazine hydrochloride) because of mild emotional problems, by her family physician. On February 22, 1964, she received 5 mg. of Prolixin by mouth, and in the late morning on February 23, 1964, she took another 5 mg. by mouth. About eight hours later (seven-thirty p.m.) she began to notice involuntary backward movement of her head, later involving her right arm and leg with arching of her back. She found she was able to overcome these movements with great difficulty and concentration. There were no sensory symptoms, and she had the presence of mind to telephone her physician. Neurologic examination was normal except for episodes of slow dystonic movements of her neck and trunk which came on in waves lasting five to ten minutes. She was apprehensive and sweaty. She was admitted to the hospital and placed on bed rest. Her symptoms subsided within six or seven hours. An electroencephalogram was done the following morning which was described as being "mildly abnormal due to disorganization of wave patterns." Laboratory studies were negative throughout, and subsequent examinations have been normal.

Case 3 (S.C. #37446): A 32-year-old female was in the hospital for investigation of other neurologic conditions and was given Compazine intramuscularly prior to and subsequent to a pneumoencephalogram. On March 4, 1964, she received 5 mg. of Compazine at noon prior to air encephalography. That evening at six p.m. she received 10 mg. of Compazine intramuscularly because of nausea and vomiting. This dose was repeated once at nine a.m. on March 5, 1964. Her nausea and vomiting subsided, but at five p.m. that day, eight hours after the last injection of Compazine, she began feeling nervous and anxious with spasms of her facial muscles and involuntary chewing movements. She was given Sodium Luminal,® 100 mg. intramuscularly, at five-thirty, and her symptoms promptly subsided, to recur once again three hours later without any additional Compazine. At eight-ten p.m., 100 mg. of Luminal were again given, and her symptoms subsided within one hour. They did not recur. The pneumoencephalogram had disclosed a mild degree of cortical atrophy.

Case 4 (D.H. #23517). A 19-year-old girl was given Combid® Spansules® (containing 10 mg. of perchlorperazine in sustained release form) because of gastrointestinal upset on October 11, 1964. That evening she took one spansule, and the following day, October 12, 1964, took three spansules. These apparently relieved her symptoms, and on October 13, 1964, she took one spansule. Between one and two hours later she began complaining of stiff neck with involuntary extension of her neck and arching of her back. She was seen in the emergency room at eleven a.m. that day, at which time she was experiencing waves of involuntary extension and rotation of her neck and back with flexion of her upper extremities. She was able to overcome these movements voluntarily with great difficulty and only momentarily. She was given 60 mg. of Sodium Luminal intramuscularly at twelve noon, two p.m., and four p.m. At five p.m., 2 mg. of Artane® were given by mouth. By nine p.m. her symptoms had entirely subsided and did not recur.

Discussion

The syndrome of reaction to phenothiazine medication is essentially that of the fairly sudden onset of uncontrollable abnormal movements, mostly of head and neck, which come upon the patient in waves, each lasting several minutes. There may be a premonition of unexplainable fear, restlessness, or both, and of tightness in the throat or neck followed by thick and difficult speech.[4] Later, contortions of the entire trunk and upper extremities, spasms of the sternocleidomastoid and trapezius muscles, protrusion of the tongue, difficulty in swallowing, and arching of the back may occur. Tremors and spasms of the upper extremities have been reported,[5] and are illustrated in one of our cases. In one report, dislocation of the jaw and chipping of the enamel of the teeth have been mentioned,[2] and laryngospasm occurred in others.[3,6] We wish to emphasize that the patients are fully awake and oriented and, as indicated in Case 2, are able to seek appropriate help. There is no loss of consciousness, no confusion, and no overt psychiatric behavior. However, uncontrollable fear and restlessness, even after the physician reassures the patient

that he will recover, is a prominent part of the syndrome.

Drugs: Similar reactions have been reported from the administration of a variety of related drugs. Chlorpromazine (Thorazine®) was implicated early in its use,[1] and perphenazine (Trilafon®) reactions are well documented.[7,8,9] Reports in the literature would seem to implicate prochlorperazine (Compazine) more frequently, and in one review the statement is made that "It seems probable that this drug (prochlorperazine) is more likely to produce these motor syndromes than chlorpromazine."[1] The present report also includes a reaction to a related phenothiazine, fluphenazine hydrochloride (Prolixin).

Dosage: Some reports suggest that greater than usual doses of phenothiazines produce a higher incidence of reaction,[4,5] while others note that small doses may well do the same.[3,4] The doses given in the present cases were all within a moderate range. In all reported cases more than one dose was given before the reaction occurred, and this was true in the cases presented here.

Onset interval with respect to last dose: The present cases illustrate that symptoms of reaction to phenothiazine may begin within several hours of the most recent administration. However, onset may be delayed more than twenty-four hours,[4] making the diagnosis more difficult. Note should be made of the obvious possible delay in onset occurring if the drug had been administered in the form of sustained release capsules.[4]

Duration: Symptoms usually last several hours but may rarely persist up to twenty-four hours. In Case 2, no specific medication was given, and the patient's symptoms persisted for six to seven hours. Appropriate medication may dramatically shorten the duration of the symptoms, but they may spontaneously recur without further administration of phenothiazine and require further treatment.

Differential Diagnosis: Differential diagnosis includes hysteria, acute schizophrenia, cerebrovascular accidents, tetanus, strychnine poisoning,[4] and unusual seizure patterns. The aware physician who keeps this syndrome in mind, and who seeks a history of phenothiazine administration, will have no difficulty with the differential diagnosis. The fact that many patients who receive these drugs are generally healthy, and particularly in the case of prochlorperazine, are undergoing symptomatic treatment while lacking the signs of other demonstrable disease, assists in limiting the differential diagnosis. There is no specific laboratory determination which will aid in the diagnosis. The quick response to parenteral sedation and the passage of time will resolve any diagnostic confusion. Unnecessary diagnostic studies such as arteriograms and trephination must be avoided unless symptoms persist for more than twenty-four hours or other neurologic abnormalities appear.

Treatment: Treatment of phenothiazine reaction consists of parenteral sedation, although cases have been reported in which meperidine hydrochloride (Demerol®), atropine, and anti-Parkinson medications have been successful.[4] Rapidly acting parenteral barbiturates appear to be the drugs of choice.[4] One reported case was successfully treated with intravenous diphenhydramine (Benadryl®).[6] This suggests that sleep induction by any means may assist in relieving symptoms until sufficient time for the reaction to subside has elapsed.[3,4,5]

Outlook: This is a benign condition. Once symptoms have subsided and the patient has recovered, the reaction will not recur unless phenothiazine is once again administered in similar doses. In some reports, patients having had a reaction to high doses of phenothiazine were able to tolerate lower doses of the same drug without return of the reaction.[2,5]

Summary

The typical phenothiazine reaction (dystonia phenothiazorum) appears to occur in young healthy individuals who are given phenothiazine for symptomatic relief of nausea or vomiting or for relief of mild anxiety. The first symptoms may be unusual apprehension and fear, followed by tight feeling in the throat and uncontrollable waves of twisting of the neck and trunk. The patient does not lose consciousness, and symptoms reach a peak within one to two hours, subsiding usually in less than twelve hours without treatment. Treatment consists of induction of sleep, most easily by administration of rapidly acting barbiturates. This is a self-limited, benign condition that responds to withdrawal of phenothiazines. This reaction is easily recognized, and elaborate diagnostic studies are not indicated.

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Dr. Chamberlin, 14 Gilman Street, Waterville, Maine
Dr. Trembly, 33 College Avenue, Waterville, Maine

A Crossroads in Medical Care

JOHN F. REYNOLDS, M.D.*

In October of last year, it was my privilege to attend a conference in Denver sponsored by the University of Colorado on the responsibilities, duties and opportunities of medical staff officers. The conference was designed for Chiefs of Staff, Chiefs of Clinical Services, and Committee Chairmen, and was attended by about 350 physicians from 43 states, the District of Columbia, 4 Canadian provinces and Puerto Rico. Five attended from Connecticut, 3 from Massachusetts, and 2 from Rhode Island. As I was the only registrant from Maine, and as all were urged to disseminate the content of the agenda, it seemed feasible to bring you some thoughts developed at the session. Without going into program detail, certain uppermost ideas should be put clearly forward for the consideration of the medical profession as a whole, and for the physicians and hospitals of Maine as part of that whole. When this session was first planned a registration of perhaps 100 was anticipated, and the response of 3½ times that number clearly indicated the need for such a program. It is hoped by those responsible to make it an annual session, and I personally can highly recommend it.

Medical and hospital history of the 1950's and 1960's will report the great phenomenon of record-breaking numbers of meetings on health, and will bear witness to the flowering of hundreds of brilliant ideas. It will also record a blight of inertia and frustration which caused great ideas and fond hopes to wither on the recording tapes. Two groups bear the primary responsibility for the improvement of hospital care in this country and they are complementary forces which must unite in a crash program for excellence. Those groups are, of course, the organized medical staffs of hospitals, and the policy-making and executive forces of governing boards and administration. Though governing boards bear legal responsibility, the structure is supported by cooperative inter-relationships of board, staff and administration. Healthy cooperation of each of the three groups, with foresight as to local problems, is imperative to success. We are privileged to live in the most enlightened era of medicine, and our medical schools and research centers progressively enrich our knowledge and develop new procedures. The American public, either through donations, fees or taxes, have provided and are providing medicine the most complete buildings and efficient equipment with which to work. All concerned, physicians, governing boards and administrators, must hold these gifts in trust for the citizens who made or are making them possible, and there is a moral responsibility to return to the citizens the best medicine and hospital care being developed today, and for which they are paying. Our riches of money, knowledge, buildings and resources contain their greatest value only when translated into services for our fellowmen. Medical progress and hospital care *are at a crossroads*. They must choose, and this rather soon, to voluntarily identify themselves with excellence, or to lapse into the category of mediocrity, dedicated to the philosophy of not rocking the boat. Physicians as a group have defaulted somewhat to the hospitals in their unwillingness to be organization members. They should and can be the greatest power in medical care, but until they take the ball and run with it, their abilities and talents will be subservient. Medicine as a whole, if organized, could change the predicted future for itself, for hospitals, and for the public, but only if it works in a way conducive to preserving its own best image. To be responsible, an individual or a group must be free, and to be free the group or individual must be responsible. The plea is made that hospitals are dedicated to serving humanity, while physicians are consecrated as it were, to administering to their fellowmen. Each group must reexamine its individual and joint roles in terms of human needs of today and in consciousness of the rich resources made available. The motivation must be the best possible care at the least possible cost.

The control of quality of medical care embraces responsibility, but goes further for it also includes the authority to carry out a program designed to achieve an objective. It has been said that the differentiation of American government vs. American freedom, is that we elect to be governed but we do not elect to be ruled. Just so, the medical profession fears control because it may mean regimentation, restrictions and imposition of rules and regulations. The doctor does recognize, however, that some controls are necessary and to his advantage, as well as to the advantage of the public, and willingly accepts these in this light. Control in some measure is more important than freedom, for if control is lost, then freedom is lost. It is time medicine paid heed to the public cause to avoid the crossroads, and greater future cooperation between physicians, governing boards and administration, each group so important to each of the others, must be the cue to achieving quality of medical care at the least possible cost.

*Chief of Surgical Service and Medical Director, Thayer Hospital, Waterville, Maine.

216 Main Street, Waterville, Maine

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Trichinosis in Aroostook County

M. C. DUNHAM, M.D.*

INTRODUCTION

Trichinosis is appearing again in Aroostook County. In the past several weeks five cases of trichinosis have occurred in a limited area of Northern Aroostook County. Prior to these cases the disease had been absent or at least unreported in this locality for several years. Now it seems worthwhile to bring these cases to the attention of the State's physicians, since, in all likelihood, additional cases are going to occur. Some infected meat from the original animal may be still "at large" in the area and perhaps, considering the extensive visiting associated with the recent holiday season, some of the meat may have been carried to other parts of the State, either before or after consumption.

CASE HISTORIES

The first cases involved two teen-age girls in a family of five. The girls prepared supper for themselves one night while the others in the family were away at camp, and in the process they cooked a two pound pork steak taken from their freezer only an hour or so earlier and therefore only partially thawed. This they cooked as they would have cooked an unfrozen steak, ie., the surface layers were cooked but the deeper layer was merely thawed. Both girls became ill, one more severely than the other. The symptoms in the case of the sicker girl began in approximately one week, consisting of transient diarrhea of 3-4 days, giving way to fever, peri-orbital swelling, marked muscle soreness, and the development of a semi-comatose state for a short period. Trichinella skin test was negative at this stage and was not repeated later. Eosinophile count reached 71%. Gastrocnemius biopsy was positive for Trichinella spiralis. The sister's illness was similar but without the initial intestinal phase.

The next two cases occurred in a man and wife who had purchased a side of beef and had requested the butcher, in preparing the hamburg portion, to grind with the 30 pounds of beef some 5 pounds of pork so that the hamburg would not be too lean. While the pair packaged the meat for the freezer, they both nibbled at the raw mixture. The husband consumed more than his wife and subsequently suffered the more severe symptoms, which included early intestinal upset followed by fever, peri-orbital swelling, weakness, and generalized soreness. Skin test in his case was positive as was gastrocnemius biopsy. His eosinophile count rose to 46%.

The fifth case involved a mother whose taste for meat differed from her children's. The family ate pork chops which were cooked moderately well. The peripheral portions were well done and this part the children ate, leaving uneaten the central parts near the bone which were still pink in color. It was this portion which the mother preferred. She finished all the rare parts the children had left, thus consuming that portion most likely to contain viable trichinae larvae. She became ill within 10 days, with fever, peri-orbital swelling, cramps and soreness of leg and arm muscles. Skin test was positive and eosinophile count reached 18%. Muscle biopsy was not done.

LIFE CYCLE

The nematode larvae of Trichinella spiralis may be present encysted in the flesh of any carnivorous mammal. The carnavore that eats larvae develops symptoms in proportion to the number of larvae it ingests. Each organism, upon release from its cyst in the intestinal tract, may develop into an adult worm. These worms produce eggs which soon develop into new larvae. This adult worm activity is responsible for the intestinal symptoms which may or may not be noticed, depending on the degree of infestation. The adult worms, after egg-laying, have completed their life cycle and soon die off, representing no significant problem in the subsequent disease.

The new generation of larvae penetrate the intestinal wall to reach the blood stream and via this to all parts of the body, but they prefer the environment of striated muscle, and there they settle out and become encysted. This causes an associated inflammation which is responsible for the swelling and soreness, varying in severity with the number and location of cysts, and for the rising eosinophile count, characteristic of parasitic infestation.

The body defense mechanisms usually respond early, resulting in

a) gradual reduction of the inflammation about the larvae;
b) formation of cyst walls within which the larvae may remain viable for years, and;
c) in 10-14 days changes in skin sensitivity and body

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fluids as shown by complement-fixation test, flocculation test and skin reaction, although all of these tests may not always be entirely reliable for diagnostic purposes.

In some cases cysts may be demonstrated in muscle biopsy.

Occasionally the infestation is overwhelming with a fatal outcome, for man or pig, but the usual course is slow recovery. Infestation is, however, more frequent than clinical cases indicate. According to various necropsy studies, one in five Americans has suffered some infestation.

Source

Pigs get their undercooked meat in the form of man's uneaten or uncooked pork left-overs put into garbage, or from eating dead infested rats, or from cannabalistic consumption of dead pigs. Obviously, the pig would not become infested if it:

a) were fed cooked garbage (required by Maine State Law);
b) were fed exclusively on grain;
c) did not have access to carcasses of rats or other pigs.

Control is difficult; the cycle is completed from time to time, and the infestation periodically recurs.

In the Aroostook cases, all five persons obtained the pork from a single local meat dealer, but in spite of investigation by the Department of Health and Welfare and the Department of Agriculture, the source of the infested pork is still unknown. The meat dealer purchased pork both locally from farmers and from western sources and is unable to state where the affected pork may have originated.

We must assume that remaining portions of the one or more infested animals have been sold and may still be in home freezers waiting to be consumed. Efforts to locate the source of the infestation will continue but are complicated by the facts that:

a) local dealers are not required to keep records as to the source of supply of pork;
b) there is no way to identify infested animals while they are alive. Thus, even if a suspect farm is located, it is not possible to determine if a given animal is affected until it is slaughtered.

Control of the disease must therefore be achieved by a program of education of the:

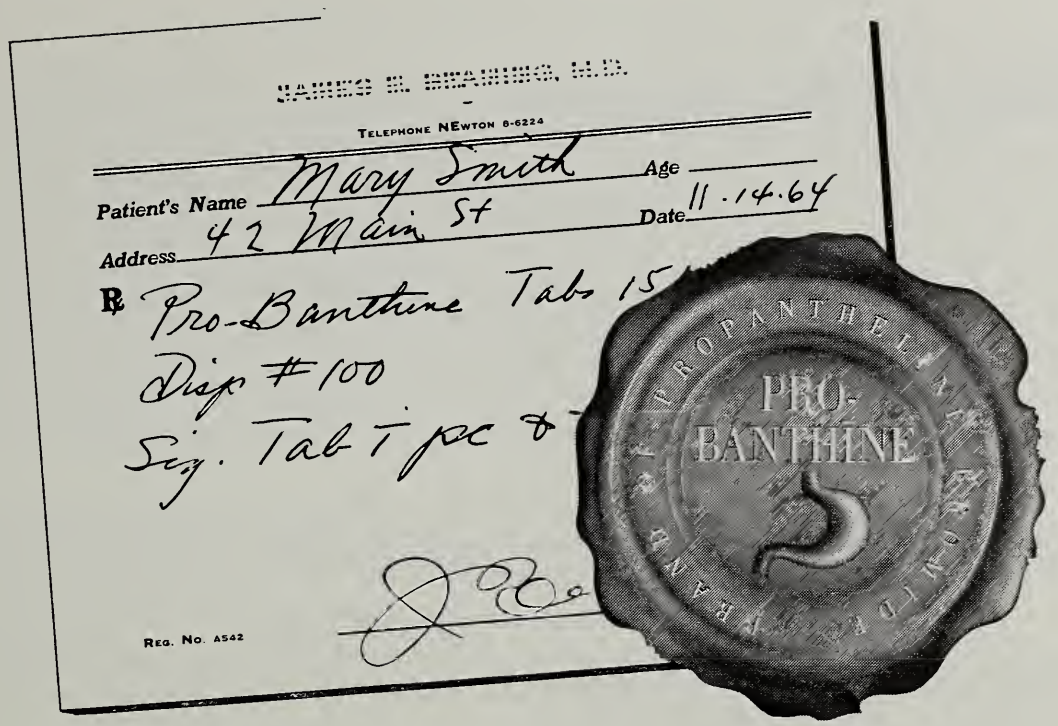
a) physician — to be alert to the possibility of trichinosis in differential diagnosis;
b) public — to cook thoroughly all pork and pork products and non-pork meat which might become contaminated by unclean butchering practices. This education should include warnings against the housewife's practice of tasting for seasoning in the preparation of home-made sausage, head cheese, and other pork products.
c) pork producers — to cook thoroughly all garbage fed to pigs. To maintain clean rat-free farms and clean, sanitary butchering practices.

Thus, although man has the knowledge to control and eradicate his age-old parasitic associate, he has yet to achieve the social skill to implement this knowledge. It would seem, therefore, that man and trichina can expect to continue their association for ages yet to come.

Annual Meeting Dates For Your 1965 Calendar

Maine Medical Association, June 13-15, 1965 at The Samoset, Rockland, Maine

American Medical Association, June 20-24, 1965 at the Americana Hotel, New York, New York



Stamp of Approval on Virtually any Ulcer Regimen-

PRO-BANTHĪNE®

(propantheline bromide)

Historically, reduction of acid and motility in peptic ulcer has been approached through the use of antacids, dietary management and surgery.

Since 1953, however, Pro-Banthīne used alone or in addition to other measures has contributed importantly to achieving both of these goals. It has been shown repeatedly that adequate doses of Pro-Banthīne will significantly inhibit gastric acid secretion and reduce gastrointestinal motility.

So dependable have these actions been that now, for many, standard treatment of peptic ulcer and several allied conditions has become antacids *plus* Pro-Banthīne, dietary management *plus* Pro-Banthīne, surgery *plus* Pro-Banthīne, or some combination of the three.

Pro-Banthīne has become the most widely prescribed anticholinergic for patients with peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia because patients respond favorably to its therapeutic actions.

Side Effects and Precautions—Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. The drug is contraindicated in patients with glaucoma or severe cardiac disease.

Dosage—The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six tablets daily in divided doses. In severe conditions as many as four tablets may be given four times daily. Pro-Banthīne (brand of propantheline bromide) is supplied as tablets of 15 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

SEARLE

Chicago, Illinois 60680

Research in the Service of Medicine

Maine Heart Association Notes

The Test Of Time

". . . of major importance today is early diagnosis because in many instances the great advances in therapeutics have become of undreamed-of importance. . . . the early diagnosis of bacterial endocarditis has meant the difference between life and death for many patients. What then are some of the symptoms and signs of the early diagnosis of bacterial endocarditis which have stood 'the test of time?'

"Patients with bacterial endocarditis usually present themselves complaining of fever, and the symptoms of a chronic infection; or symptoms resulting from embolism to the peripheral arterioles of the skin, brain, spleen, or kidneys.

"In the physical examination the important signs are: (1) fever associated with a valvular heart murmur; most often a mitral or aortic valvular murmur, (2) hemorrhages into the conjunctivae, skin, under the nails, (3) clubbing of the fingers and (4) splenomegaly.

"The significant points in the history are: (1) rheumatic fever or congenital heart disease in the past, and (2) a recent respiratory infection, tooth extraction, pregnancy or surgical operation, especially upon the urinary tract.

"The most important laboratory test is the blood culture, and when positive and associated with a valvular murmur and any of the above symptoms, the diagnosis is practically certain.

". . . cultures are negative in fifteen to twenty-five per cent of cases.

"When bacterial endocarditis is present without positive blood cultures, the patients . . . that give rise to difficulty in early diagnosis are those without cardiac murmurs or without fever when they are seen for the first time.

"These patients usually present themselves with signs of a neurological disorder, either aphasia, hemiplegia, cranial-nerve palsies, or signs of acute glomerulonephritis. . . .

"The patients who present themselves with signs of glomerulonephritis are likely to have negative blood cultures.

"Bacterial endocarditis should be considered as a diagnostic possibility when a young adult complains of drowsiness, severe headaches, double vision, impairment of speech, or loss of consciousness, particularly when he shows cranial-nerve palsies or a hemiplegia.

"These neurological manifestations may be the earliest evidence of the existence of bacterial endocarditis and may occur before fever or before cardiac murmurs are recognized.

"It is important then for all physicians to be familiar with the early signs . . . so that an early diagnosis can be considered and treatment started.

". . . Early and precise diagnosis based upon symptoms and signs which have withstood the 'test of time' calls for therapeutic action."

Reference: Keefer, Chester S. The Boston Medical Quarterly, Volume 15, pages 3-5, 1964.
Submitted by Jacob B. Dana, M.D.

County Society Notes

ANDROSCOGGIN

A meeting of the Androscoggin County Medical Society was held at the Central Maine General Hospital in Lewiston, Maine on October 22, 1964. The meeting was called to order by the President, Robert D. Wakefield, M.D.

Ralph A. Goodwin, Sr., M.D. reported for the resolutions committee on the death of Wallace E. Webber, M.D. It was voted to send a copy to the family, the Maine Medical Association and to the Executive Director of the Central Maine General Hospital.

Michael J. Harkins, M.D. brought up for discussion the changing of voluntary contribution to an assessment without the society being informed prior to the June meeting of the House of Delegates. A motion was made to table this until the December meeting. (This discussion to be printed on notices.)

A nominating committee, consisting of Drs. Paul J. LaFlamme, Chairman, Lawrence A. Nadeau and Frederick B. Lidstone, was appointed by the President to prepare a proposed slate of officers for 1965.

An interesting film, Part II, Stroke and Rehabilitation was shown. This depicted early prodromal signs, occurrence of the stroke with immediate then later remedial therapy. The role a family could play in rehabilitation was stressed. Practical physiotherapy apparatus and exercises both active and passive were integrated into recovery. Emotional problems were demonstrated and explained.

DONALD L. ANDERSON, M.D.
Secretary

PENOBSCOT

A meeting of the Penobscot County Medical Society was held on November 17, 1964 at the Millinocket Community Hospital. The President, William A. Purinton, M.D. presided.

Francis Woods, M.D. from the Overholt Clinic gave an interesting talk on X-ray Problems in the Diagnosis of Chest Cancer.

The following slate of officers were elected for 1965:
President, Richard T. Munce, M.D., Bangor
Vice-President, Herbert C. Gilman, M.D., Millinocket
Secretary, Charles D. McEvoy, Jr., M.D., Bangor
Treasurer, Benjamin L. Shapero, M.D., Bangor
Executive Committee, Drs. George W. Wood, III, Brewer (2 yrs.) and Leonard G. Miragliuolo, Bangor (3 yrs.)

HADLEY PARROT, M.D.
Secretary

CUMBERLAND

The Cumberland County Medical Society meeting was held on November 19, 1964 at Valle's Steak House in Portland, Maine with 81 members and guests attending.

After a social hour and dinner, Albert Aranson, M.D. reported that the Health Insurance Committee had recommended that payment for care of veterans by private physicians be made according to the California relative value scale, with a $5.00 medical unit and a $350.00 surgical schedule, similar to the BSC contract. He also announced a federal employee's $7,500.00 program to include more employees under service benefits. In response to questions, both Dr. Aranson and Dr. Geer emphasized that the BSB Blue Shield contract is no longer being sold but is still maintained by some policy holders.

Boris A. Vanadzin, M.D. reviewed the work of the Health Careers Committee in 1964 and stated his recommendations for the ensuing year. The question of including college freshman in the program was brought up but not fully discussed. Dr. Robert McAfee reported on his activities with the high school pre-medical clubs.

A nominating committee, consisting of Drs. Sidney R. Branson, Chairman, Alvin A. Morrison and Charles R. Glassmire, was appointed by the President.

Wilbur F. Leighton, M.D. discussed the duties of the medical examiner as prescribed by law, emphasizing that the problem areas involve the sudden and unattended deaths. Dr. Leighton introduced Mr. Franklin H. Stearns, Jr., the County Attorney, who suggested that many murders are probably passed off as sudden death due to natural causes. He stressed the fact that murder is usually a "family affair" and that if there is any question about the cause of death, the physician should call the medical examiner to protect himself from potential unfavorable publicity.

G. Hermann Derry, M.D. pleaded for recognition of the "Battered Child" syndrome and for support of a law requiring reporting of suspected cases to the medical examiner.

After discussion of Medical-legal experiences by Dr. Fagone and Dr. Freeman the meeting was adjourned.

A meeting of the Cumberland County Medical Society was held on December 17, 1964 at Valle's Steak House in Portland, Maine with 73 members attending.

After a social hour and dinner, Paul S. Hill, M.D. of Saco, Councilor for the First District, was introduced. The secretary read a letter from Mr. Robert W. O'Connor regarding the current legislative program and it was decided to send a copy of Mr. O'Connor's letter to each member of the society.

Howard P. Sawyer, Jr., M.D. stated that one effective way of influencing legislators is to testify at committee hearings. William L. MacVane, Jr., M.D. recommended mobilization of paramedical health groups.

Payson B. Jacobson, M.D. asked the secretary to find out from Mr. O'Connor the outcome of the two optometric bills in the last legislature and what action should be taken in the future in regard to such bills should they come up again.

Sidney R. Branson, M.D., Chairman of the Nominating Committee, presented a slate of officers for the year 1965 which was unanimously approved.

President, Alvin A. Morrison, M.D., Portland
Vice-President, Charles W. Capron, M.D., Portland
Secretary-Treasurer, Stanley B. Sylvester, M.D., Portland
Delegates to the Maine Medical Association House of Delegates: Drs. Charles R. Geer, Howard P. Sawyer, Jr., Maurice VanLonkhuyzen, Philip P. Thompson, Jr., David K. Lovely and Clement A. Hiebert, all of Portland (2 yrs.); Drs. Robinson L. Bidwell, John F. Gibbons, Charles R. Glassmire and Donald P. Cole, all of Portland (1 yr.). Alternates: Drs. Hugh P. Robinson, Alfred W. Hurwitz, Irving J. Poliner, Louis A. Ciampi, all of Portland, Raphael F. Turgeon, Westbrook and Harold N. Burnham, Gorham (2 yrs.); Drs. Lloyd G. Davies, Fryeburg, A. Dewey Richards, Bridgton, Stanley W. Kent and George F. Sager, both of Portland (1 yr.)

Dr. Sawyer reported on the Legislative Committee and stressed three points:

1. That the AMA opposition to Medicare will be tailored on an individual state basis.
2. That the medical profession should try to convince the public that Kerr-Mills is working.
3. That the auxiliary should be utilized to inform the public.

Dr. Charles R. Geer read the report of the Public Relations Committee as prepared by Eugene C. McCann, M.D. Dr. Geer

appointed a committee to audit the societies books, to consist of Drs. George I. Geer, Jr., Chairman, Francis H. Fox and William J. White.

It was voted to waive any charge for dinners for the non-dues-paying members of the society.

It was voted to write to the City Council of the City of Portland urging them to follow the recommendations of the United Community Services' Hospital Planning Committee when undertaking a building program at the Portland City Hospital.

Dr. Charles R. Geer delivered an address upon completion of his term of office prior to turning over the gavel to Dr. Morrison who adjourned the meeting.

STANLEY B. SYLVESTER, M.D.
Secretary

KNOX

The Knox County Medical Society meeting was held on December 1, 1964 at the K of C Hall.

The following officers were elected for 1965:

President, Frank W. Kibbe, M.D., Lincolnville
Vice-President, Onni C. Kangas, M.D., Rockland
Secretary-Treasurer, Peter H. Holz, M.D., Camden
Delegates to the Maine Medical Association House of Delegates: Drs. Johan Brouwer, Rockland and Harry G. Tounge, Jr., Camden. Alternate Delegates: Drs. Wesley N. Wasgatt and William W. Ward, both of Rockland

ONNI C. KANGAS, M.D.
Secretary

HANCOCK

A meeting of the Hancock County Medical Society was held on December 9, 1964 at the Hancock House in Ellsworth, Maine.

A letter from Mr. Robert W. O'Connor of Augusta regarding the current legislative program was read to the society.

The following officers were elected for 1965:

President, W. Edward Thegen, M.D., Bucksport
Vice-President, Raymond E. Weymouth, M.D., Bar Harbor
Secretary-Treasurer, Russell G. Williamson, M.D., Blue Hill
Delegates to the Maine Medical Association House of Delegates: Drs. Russell M. Lane, Blue Hill and Herbert T. Wilbur, Jr., Southwest Harbor. Alternate Delegates: Drs. Llewellyn W. Cooper, Bar Harbor and Philip L. Gray, Blue Hill
Censors: Drs. John D. McIntyre, Blue Hill (3 yrs.), W. Edward Thegen (2 yrs.) and Arthur M. Joost, Jr., Bucksport (1 yr.)

The Hancock County Medical Society held a meeting at the Hancock House in Ellsworth, Maine on January 13, 1965.

Harold D. Cross, M.D. of Hampden Highlands was the guest speaker. He spoke on Hypnotism in General Practice. Mason Trowbridge, Jr., M.D. of Bangor acted as a guest subject and presented appropriate comments on the subject of hypnotism.

RUSSELL G. WILLIAMSON, M.D.
Secretary

KENNEBEC

The Kennebec County Medical Society meeting was held on December 19, 1964 at the Augusta State Hospital in Augusta, Maine. The meeting was called to order by the President, Kenneth W. Sewall, M.D.

Brinton T. Darlington, M.D. noted that our legislative representative, Mr. Robert W. O'Connor of Augusta has called attention to the fact that some of the legislature which was defeated in previous years will be introduced this year.

Thomas A. Martin, M.D., President of the Maine Medical Association, was introduced and addressed the meeting briefly.

Philip Dachslager, M.D., Chairman of the Nominating Committee, presented a slate of officers for 1965 which was approved:

President, John D. Denison, M.D., Gardiner
Vice-President, Allan J. Stinchfield, M.D., Augusta
Secretary-Treasurer, Earle M. Davis, M.D., Waterville
Delegates to the Maine Medical Association House of Delegates: Drs. Kenneth W. Sewall, Valentine J. Moore, Paul A. Jones, Jr., all of Waterville, William N. Runyon, Augusta and Richard E. Barron, Monmouth. Alternates: Drs. Francis J. O'Connor and John M. H. Barnard, both of Augusta, Richard T. Chamberlin and Richard L. Chasse, both of Waterville and Robert H. Dunn, Togus
Councilors, Drs. Robert L. Ohler, Togus, Richard H. Dennis, Waterville and Joseph R. Crawford, Augusta
Grievance Committee, Drs. Oakley A. Melendy, Chairman, Augusta and Edmund N. Ervin, Waterville
Man Power Procurement and Civil Defense Committee, Drs. Albert A. Poulin, Chairman, Waterville, M. Tieche Shelton, Augusta and Clarence R. McLaughlin, Gardiner

Dr. Sewall introduced Alta Ashley, M.D. of the Department of Health and Welfare who in turn introduced Mr. Robert Wyllie, a medical unit director of the Department of Health and Welfare. Mr. Wyllie spoke relative to the Kerr-Mills Law and Medical Aid to the Aged here in the State of Maine. Mr. Wyllie made the point that the law which was passed in 1960, amending the Social Security Act was designed to help those over 65 who are usually independent economically but who are unable to care for high medical expenses. He illustrated the method for medical aid to the aged and enumerated the services provided under law. There were several questions from the floor and Dr. Darlington elaborated on the difficulties met within the State of Maine with Federal matching funds as now available.

EARLE M. DAVIS, M.D.
Secretary

New Members

AROOSTOOK

Alroy A. Chow, M.D., Main St., Ashland
Necdet Gureli, M.D., Cary Mem. Hosp., Caribou

HANCOCK

Horace B. Pease, M.D., Maine Coast Mem. Hosp., Ellsworth
Eliot T. Stadler, M.D., West Gouldsboro
Robert D. Wilson, M.D., Mt. Desert Island Hosp., Bar Harbor

KENNEBEC

Demitrios Nikolaidis, M.D., Veterans Adm., Togus

KNOX

Sallie H. Saunders, M.D., Route 1, Lincolnville

YORK

Willem F. Nieuwkerk, M.D., P.O. Box 424, Kennebunkport
Harold W. Stevens, M.D., 369 Ferry Rd., Saco

News, Notes and Announcements

Postgraduate Course in Clinical Cardiology
Central Maine General Hospital
Lewiston, Maine

Co-sponsored by the
Central Maine General Medical Staff and
The Maine Heart Association, Inc.

Hiebert Hall 3:00-5:30 P.M.

March 3, 1965 "Bedside Examination of the Heart"
Herbert J. Levine, M.D.

March 10, 1965 "Congestive Heart Failure"
Roman W. DeSanctis, M.D.

March 17, 1965 "Congenital Heart Disease"
Walter Goodale, M.D.

March 24, 1965 "Diagnosis and Treatment of Cardiac Arrhythmias"
Lawrence Ellis, M.D.

March 31, 1965 "Recognition and Management of Coronary Heart Disease"
David Littmann, M.D.

Fee: $25.00.
No fee for interns or residents.

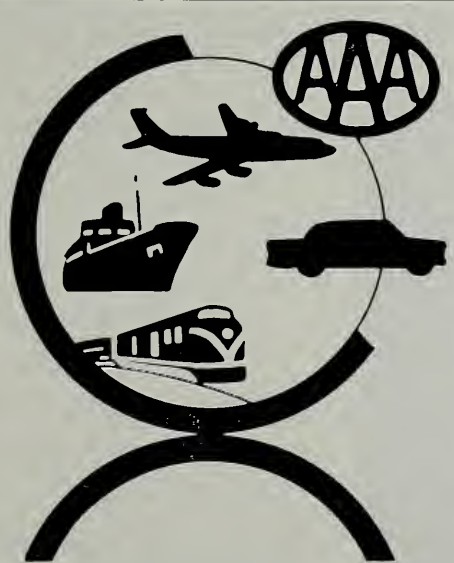
Seminar in Prosthetics

Orthopedic surgeons, general surgeons, specialists in physiatry, and others interested in problems of the amputees are urged to attend a seminar concerned with this area at the Holiday Inn, Auburn, on Wednesday, April 7th.

The seminar will be sponsored by the Vocational Rehabilitation Administration through an in-service training grant to the Maine Division of Vocational Rehabilitation. A team of specialists will discuss amputation surgery and postoperative care, prosthetics and recent advances made in the appliance field, and the vocational rehabilitation of amputees. There will also be a live demonstration of the amputee clinic team in action.

The tentative program for this seminar is as follows:

9:30- 9:45 A.M. — Opening of the Conference
9:45-10:30 A.M. — "Limb Prosthetics Today"
Dr. H. W. Glattly
10:30-10:45 A.M. — Coffee Break
10:45-11:30 A.M. — "Amputation Surgery — Postoperative Care"
Dr. Eugene E. Record
11:45-12:15 P.M. — "Recent Advances in Prosthetic Appliances — Present Research Program"
Mr. Ivan A. Dillee
12:30- 1:45 P.M. — Luncheon
1:45- 3:15 P.M. — "Amputee Clinic Team — Demonstration of Patients"
Dr. Roy M. Hoover
Mr. Fred Murko
3:15- 3:45 P.M. — "Vocational Rehabilitation of the Amputee"
Dr. Bernice Clark
3:45 P.M. — Closing Discussion



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The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, March, 1965 | No. 3

Boston Medical Reports

A Postgraduate Educational Television Series For Practicing Physicians In The State Of Maine, A Preliminary Review And Report Of Initial Evaluation

George J. Robertson, M.D.,* Harold F. Pyke, Jr.** and Edward M. Friedlander***

In the report on *Medical Care In The State of Maine, 1956-1962,*[1] Dr. Jean A. Curran, Senior Consultant of the Bingham Associates Fund, summarized Chapter X, entitled "Continuing Education of the Physician" as follows:

> "The record of continuing education of physicians in Maine reveals very encouraging advances and new developments, but much remains to be done in fostering and utilizing in every way possible the newer methods of communication which can help to keep the practitioner current with the latest developments in his fields."

During the past year, television has been utilized as one of "the newer methods" to which Dr. Curran referred. How this developed and was carried out, and some preliminary measure of the effectiveness of this medium based on studies already done, can now be reviewed.

In 1963, the year following the publication of the aforementioned report, Dr. Curran, George T. Nilson, Bingham Associates Fund Field Director, and Harold F. Pyke, then a member of the staff of the Boston Educational Television Channel WGBH-TV, conducted a study in the State of Maine to establish the feasibility of using television as a postgraduate educational medium there. Their study revealed that Maine physicians were sincerely interested in this medium as a possibility for a continuing educational program. Also established by the study was the fact that television coverage of the state could be accomplished through the use of the state's Educational Television Network of three existing channels, a fourth near completion, and a fifth located in nearby Durham, New Hampshire, which also serves an adjacent section of Maine.

Following negotiations with officials of both WGBH-TV and the Postgraduate Medical Institute, the educational division of the Massachusetts Medical Society, an initial series of 12 presentations was planned. Entitled "Boston Medical Reports," subjects for the series were selected on the basis of their interest to general practitioners and internists by a Program Committee established for this purpose. In addition to the Executive Director of the Maine Medical Association and the Medical Director of the Bingham Associates Fund, the committee included the director and members of the Program Committee of the Postgraduate Medical Institute.

Selected to present the subjects chosen were members of the faculties and staffs of Boston's three medical schools and their affiliated teaching hospitals. These choices were made on the basis of not only their familiarity with the subject to be covered, but on their recognized abilities to present their material clearly and concisely before an audience.

The format of the programs was designed to give the presentors 30 minutes in which to cover their material. It should be noted that some prior discussion and preparation of both the material to be used and the manner in which it was to be presented was required. This was

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done in the hope of achieving as comprehensive a coverage of the subject as possible and the maintenance of interest by the viewers. Having announced the series in advance and invited questions on the topics to be presented, an additional 10-minute period was added to the program in which selected questions which had been submited could be answered.

All programs were taped by WGBH-TV during January, February and March of 1964 in a specially equipped room in the Pratt Clinic-New England Center Hospital at the Tufts-New England Medical Center. These were subsequently televised each Tuesday at 10:20 p.m. for 12 weeks starting on March 3. This particular time was set so that the program could start five minutes after the regular evening programming had been completed, and because it was expected that at that time the physician would most likely be at home and able to watch.

It should be noted that since educational television is open circuit and therefore available to anyone with a standard television receiver, viewing of "Boston Medical Reports" could not be limited to physicians alone. This accounted for scheduling the programs five minutes after the close of the regular programming. No public announcements were made of the programs, and during the entire 12-week period no single difficulty ensued. Therefore, it was concluded that members of the lay public either did not watch these programs or, if they did, were apparently not interested.

As a result of the efforts of the Postgraduate Medical Institute, each program in the series was presented one week later in Massachusetts via WGBH-TV and the Eastern Education Network. In this case it was shown each Monday at noon. In addition to this being a time not normally scheduled for regular programming by the channels, it was felt that physicians could and would watch at locations in their own hospitals.

Since television has been used in several other areas of the country as an educational medium for physicians for a number of years, an evaluation of the various efforts is of concern and interest to all who are involved in this field.

During the initial planning stage and prior to the actual presentation of the series, advice from consultants in evaluation techniques was solicited and followed.

Their first recommendation was to direct as much effort as possible to the objective of gaining and maintaining an audience. A review of other similar programs indicated that only 10 to 20% of the total potential audience could be expected.[2,3] Because of the limited number of physicians in the State of Maine, the cost would be prohibitive if only this percentage of viewers was realized. To avoid this, they recommend that a well-conceived and well-executed public relations and promotional effort be directed toward the total potential audience.

To achieve this end, a series of attention-getting mailings were prepared and carried out to all physicians in the State of Maine. In addition to attractive and informative material, first announcing the series, its purposes and its format, and then providing background material for the individual programs, a three-ring notebook imprinted as "Physician's Notebook for Boston Medical Reports," was provided to all physicians in which they might keep all program materials. Also included in the notebook were blank pages where notes could be taken by the viewing physician during each program. Subsequent mailings were all prepared and punched with three holes to fit into these notebooks. The purpose for providing these materials was not only to encourage a maximum viewing and participating audience, but to enable that same audience to draw as much as possible from the programs and maintain it as a reference for the future.

In this connection, the consultants on evaluation techniques recommended that testing for knowledge acquired or attitude or behavioral changes in practice among physicians should not be attempted in connection with the initial series, but left to a future series. Hopefully this can be reported in a subsequent paper.

The evaluation of the initial series in terms of viewing audiences of various programs, and a measurement of the value and degree of interest those programs had, was carried out by Dr. Ezra Saul and Mr. Richard Carter of the Department of Psychologic Research at Tufts University.

Three methods of evaluation were used. These included (1) postpaid reply cards for each program made available with program material; (2) opinions of the total series expressed on question and answer cards distributed at the Annual Meeting of the Maine Medical Association in June; and (3) personal interviews with a representative group of Maine physicians both at the Annual Meeting and during the summer months that followed.

The mail reply card analysis revealed a fairly good return for the first few programs, however this gradually diminished with subsequent programs. The diminishing number can be attributed to several factors that were substantiated by comments made during subsequent personal interviews. First, many physicians neglected to fill out and mail cards after the novelty of expressing their opinions had worn off following the first few programs. Secondly, the last few programs of the series were presented in the late spring when many physicians were becoming involved in society meetings, conventions, etc. If they did see the programs, they saw them on the run; but the fact is that a lesser number of physicians did watch these later programs.

Of the 284 physicians registered at the Annual Meeting of the Maine Medical Association in June of 1964, 208, or 79% of the total, completed and returned the "Boston Medical Reports" question and answer cards during the meeting. From the information provided by

these cards, it was calculated that an average of 3.34 of the 12 programs were seen by this particular group of physicians.

During the same meeting 68, or 25% of those registered, submitted to a personal interview of from 8 to 12 minutes duration. Using the same interview format, an additional 89 physicians were personally interviewed during the summer of 1964. Thus, a total random sampling of 157 physicians were questioned individually.

A preliminary analysis of the results of these interviews reveals that of the total audience potential of 735 physicians actively practicing in the State of Maine, an average of 30.6%, or 225, watched each program. In addition, 13.4%, or 99, read some of the reference material provided for each program. Projecting the information provided indicated that 71.8% of all physicians who viewed any part of the series were enthusiastic and described the programs as interesting, informative, and professionally useful to them.

It would appear from these figures that audiences of significant size were achieved and maintained for all programs, and that these audiences had a sufficiently deep interest to justify the initial effort and expense.

The financial aspects of the series are of some interest. Figures indicate that the total cost of each program was about $4,000, or slightly more than $5 per potential viewer, and slightly less than $20 per *actual* viewer in the State of Maine. This formidable cost, plus additional time and effort otherwise expended, demands thoughtful consideration. However, these considerations can be tempered by the fact that, although there are currently no figures available of the number of Massachusetts physicians who watched the series, the cost per physician actually watching each program would drop considerably. In addition, the fact that the tapes of some of the programs have been, and will continue to be, made available to other outlets will further decrease the per viewer cost. Further, the initial steps that were taken, the apparent quality of the programs, and the favorable reaction that was attained, all combined to help realize assistance for further programs and series from the U. S. Public Health Service in the form of a three-year grant to the Bingham Associates Fund.

The initial results of the study among Maine physicians have also contributed much helpful information which has already been applied to the 1964-1965 series. The Television Committee of the Maine Medical Association has been most helpful with constructive suggestions, and the critiques presented in *The Journal of the Maine Medical Association*[4] by Dr. Mason Trowbridge have humbled us. While the same group of consultants are planning new evaluation procedures to measure the current series, we hope for continued cooperation and criticism by and from Maine physicians.

Summary

"Boston Medical Reports" is a medical postgraduate educational television series planned, produced, taped, promoted and broadcast via open circuit television to Maine physicians. The first series of 12 programs during 1964 was developed as the result of a feasibility study conducted in Maine by the Bingham Associates Fund. How a sufficiently large and interested viewing audience was attained to justify the effort and expense is described. Some measure of both the number of viewers and their reaction to the series is also detailed as the result of evaluations made by professional consultants in this field. A second series is currently in progress, and further studies of this type of approach as a postgraduate educational medium seems warranted.

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ANNUAL MEETING DATES FOR YOUR 1965 CALENDAR

Maine Medical Association, June 13-15, 1965 at The Samoset, Rockland, Maine

American Medical Association, June 20-24, 1965 at the Americana Hotel, New York City

Progeria in the Adult*

H. S. Baar, M.D.** and J. Galindo, M.D.***

Hutchinson in 1886,[42] under the title of "Congenital Absence of Hair and Mammary Glands" was the first to describe the clinical characteristics of Progeria. In this description the boy had, in addition to the signs mentioned in the heading, an atrophic condition of the skin and its appendages and his mother was almost completely bald from alopecia areata since the age of six. The term "progeria" was, however, introduced in 1904 by Gilford[34] to designate a condition characterized by low stature, senile changes, and infantilism combined, "They pass from delayed childhood to premature old age." Variot and Pironneau[113] and other French physicians have preferred the term "nanism type senile" or "nanism progerique" (Roussel et al.[93]) A total of 23 typical cases of this disease had been reported in the world literature until 1951 (Rossi[91]) and in 1963 Djupesland[22] collected 35 examples of this disorder. Our own survey of the literature revealed 39 cases.[1,3,10,13,16,18,19,22,24,26,29,30,32,33,34,38,42,43,50,53,58,61,62,66,67,70,71,73,74,79,80,82,84,86,90,91,93,97,98,99,101,105,107,108,111,112,120,122,123] One case was studied at the Birmingham Children's Hospital (England) and one of the writers (H.S.B.) had the opportunity to examine the histological preparations. The case was used at Professor van Creveld's Paediatric Clinic in Amsterdam as basis for a doctor thesis and may be included in one of the Dutch papers.

The clinical picture of this affliction is very characteristic. The patient appears as an old, undeveloped, usually, except for the relative shortness of lower extremities, well-proportioned dwarf; thin, with dry, wrinkled, and atrophic skin, scanty subcutaneous tissue, and prominent superficial veins, particularly on the head. Zeder[122] compares the appearance with that of a "very old cobold," not larger than an infant. The hair of the capillitium is very scanty or, in fact, is totally absent; on occasions, it consists of only a few gray hairs on the scalp. The mandible is usually receding and is poorly developed and, due to this smallness, the teeth show malocclusion. Since the eye-slits are widened as a result of atrophic skin changes in the eyelids, the eyes appear prominent and buldging. There is a disparity between cranium and face, the latter appearing camparatively small. The extremities are thin, the lower usually shorter than the upper. The nails are seen undergoing atrophic changes, becoming brittle, dry, and excessively wrinkled. The external genitalia are underdeveloped, and although the pubic hair is absent, a somewhat thickened panniculus adiposus is often encountered overlying the pubic symphysis. The long bones may show a broad epiphyseal line and the distal phalanges are short. The shortness of distal parts of extremities has been designated as acromicria contrasted with acromegaly and considered as evidence of pituitary hypofunction (Strunz[107]). The cranial bones are thin and may show conspicuous impressiones digitatae. Coxa valga is frequently encountered and the patients may show the so-called "Horseman's Stance." The clavicles are short (this was studied by Legre et al[59]) and the chest is more conical than usual. The shoulders are rounded and narrow. Quite frequently, patients suffering from progeria have normal or above normal intelligence and the facial expression, with wrinkling of the forehead, slitting of the eyes, the beaked nose, the receding chin, gives the impression of a wizened, old person. Moehlig,[66] however, described a case of an otherwise typical progeria with low mentality and congenital cataracts. All patients with progeria show a striking resemblance of appearance, amounting almost to identity, which suggests a disease entity and makes it easy to separate cases of real progeria from those resembling progeria or "progeroid." The resemblance is so spectacular that the father of Gilford's patient mistook the photograph of another progeria-child for that of his own son.

X-ray examination of the bones may reveal generalized osteoporotic changes of a moderate degree. The disease usually makes its appearance at the end of the first year of life and is characterized by a gradual slowing down of growth and progressive loss of hair, thinning of subcutaneous tissues, and all the previously enumerated changes. The growth curve[18] shows not only a marked reduction in the velocity but also the absence of the "hump" characteristic for puberty. All facets of this disorder may make a simultaneous appearance or may show disparity in chronilogical sequence. With increase in age and progression of the disease, the patients are frequently affected by arteriosclerosis which may terminate fatally in cerebral vascular accidents, coronary thrombosis, or hypertensive states. Keay et al[52] described a pattern of serum-lipids and lipoproteins similar to that seen in coronary sclerosis. The disease appears to be a recessive hereditary disorder, as pointed out by Paterson in 1922[82] and by Mostafa and Gabr in 1954.[70]

Chromosomal studies were carried out in two cases (Moynahan,[71] and Djupesland.[22] The chromosome number and the karyotype were normal in both instances.

There are a number of reports on a condition which resembles more or less progeria and which has been labelled "Progeria in the Adult" or "Werner's Syn-

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***Director of Pathology, Pineland Hospital and Training Center, Pownal, Maine.

drome," the two designations being used as synonymous. But, while the appearance of Hutchinson-Gilford's progeria is so characteristic that there is no difficulty in listing doubtless cases reported in the literature, the opinions as to what is to be considered as Werner's syndrome are so variable that a reliable list of reported cases is almost impossible. Knoth et al[54] in 1963 listed 65 publications with usually one, sometimes 2-4, reported cases. The list includes all four of Werner's cases, of which only one was dwarfed. In other cases, the height is not mentioned at all. Werner described his cases under the heading "Cataract in Association with Scleroderma," but subsequent authors have, in a variable way, enlarged on the syndrome. Thannhauser[110] lists the following symptomatology of Werner's syndrome: 1. Short stature and characteristic habitus, 2. Premature graying of hair, 3. Premature baldness, 4. Scleropoikiloderma, 5. Trophic ulcers of legs, 6. Juvenile cataracts, 7. Hypogonadism, 8. Tendency to diabetes, 9. Calcification of blood vessels, 10. Osteoporosis, 11. Metastatic calcification, 12. Familial tendency. Matras and Kohler[63] consider a triad characteristic of Werner's syndrome, namely: 1. "Tight atrophy" of skin with development of ulcers, 2. Bilateral cataract, and 3. Presence of endocrine disturbances (hypogenitalism and diabetes mellitus). Several authors consider dwarfism or at least small stature as obligatory, while in the literature, the height varies between 100 and 167 cm. Rud[94] calls a patient of 160 cm. height "small in stature" and one of Krebs, Hartmann, and Thiebaut[55] had a height of 167 cm. Ten per cent of all cases described as Werner's syndrome developed malignant growth (Bau-Prussak[6]). For the majority of patients who deserve the label "Werner's syndrome," it can be said that it consists of juvenile cataracts, atrophic skin changes with ulcers, short stature, familial occurrence. The syndrome has been confused with Rothmund's syndrome. The differences between the two syndromes have been clearly shown by Thannhauser[110] and later by Greither.[39] The latter pointed out that "the names acromicria and progeria should not be used for the Werner-syndrome (for the Rothmund-syndrome they are still less suitable)." If only the complete disorder is considered, Werner's syndrome may be described as follows: The first sign is usually graying and thinning of scalp hair in the third decade of life. The skin becomes taut, thickened, which results in difficulties in walking because of the fixation of joints, and is associated with chronic indolent ulceration. Cataracts make their appearance early in life. The patients are short of stature and may show any or all of the following symptoms; hypogonadism, calcification of blood vessels, often a tendency to diabetes mellitus, osteoporosis and metastatic calcification. The voice is usually high-pitched, and the vocal cords may show a peculiar picture of irregular white areas which vary considerably in size and shape and are separated from each other by interlacing bands of reddened mucosa. The affected areas of the skin are usually most prominent on the lower extremities, but do not usually confine themselves to that region. The patients have usually a protuberant abdomen and a stocky thorax. Oppenheimer and Kugel[77,78] were, in 1934, the first to report this syndrome in American literature and suggested to replace the name "Progeria of the Adult" by "Werner's Syndrome." Later, they reported post mortem findings. In addition to the previously mentioned symptoms, their patients had blue sclerae and one of the patients had a positive Hamilton test. This patient had also a carcinoma of the liver and an adenoma of one adrenal. They assumed an excess of circulating parathormone, but the association appears to be fortuitous. The inheritance of Werner's syndrome appears to be recessive or irregularly dominant. The disorder was described occurring in four siblings, two males, 36 and 40 years old, and two females, 36 and 38 years of age. The fifth sibling was healthy. Cases showing only dermal changes and cataracts or premature balding or premature graying have been described with questionable justification as formes frustes. Occasionally, cases are observed which show precocious cataract, "Scleroderma" and infantilism without signs of senility (Guillain et al[41]). In another case, total alopecia was observed with trophic changes in the nails (Papastratigakis [81]). Some authors describe a syndrome similar to that of Werner but without cataracts as "degenerescence genito-sclerodermique" (Bau-Prussak[6]) or "gerodermatolisia genito-dystrophica" (de Toni et al[21]).

Another, but unrelated, syndrome which is sometimes called "progeria-like" or "progeroid" is cutis marmorata congenita teleangiectatica (Wieser[117]; Terrien and Prelat[109]). Among the papers dealing with progeria-like syndromes that of Bamater et al[4] should be mentioned. These authors reported on a case of hereditary geroderma and stressed that, although there is some similarity to progeria, the two conditions should be separated.

The purpose of this paper is to describe the case of an adult who shows almost all symptoms of Hutchinson-Gilford's progeria, including a peculiar facial appearance, to report metabolic and endocrinological studies, the chromosomal counts and karyotype and to compare, in the form of tables, the symptomatology of progeria, Werner's syndrome, and our case. Finally, a hypothesis will be put forward in which progeria is considered as generalized abiotrophy of multifactorial genetic origin.

Case Report

L. M., born January 14, 1920, was admitted to the Pineland Hospital and Training Center at the age of 17 years and 6 months, the daughter of a laborer in good health who was partially bald since the age of 28 years. The mother was healthy, but rather short-statured (5 feet, 5 inches=164.5 cm.). The grandparents, both on the paternal and maternal side, were normal. The family consisted of 16 siblings, of which our patient was the fourth in order of delivery. All siblings were physically and mentally normal. All attended school, although none of them finished high school education. There was no consanguinity of parents or grandparents. L. M. was delivered at home, a full-term female infant with the weight of



FIG. 1. Anterior and lateral views, showing the senile appearance, wrinkled skin, beaked nose, short phalanges, "Horseman's stance," flat, atrophic breasts, coarse hair.

only 2½ pounds (=1.132 kg.). During the pregnancy the mother continued to menstruate regularly during the first 6 months, after which she bled no longer until just prior to and during delivery. The baby, at birth, showed a more wrinkled skin than the other children in the family. She showed no additional abnormalities except of the fingernails and toenails, which were wider than they were long, and very soft. The mother noticed that the baby's breasts were somewhat enlarged, but when she pressed them she obtained no discharge. Both parents relate that the infant was so small that a shoe box had to be used for her in place of a crib. Although the infant had a normal appetite, she progressed slowly in stature and in weight. The teeth erupted on time, but showed wide variation in size of the interdental spaces, and in places there was overriding by other teeth. Vocalization of sound occurred at the age of 3 or 4 months, but throughout her lifetime she has only learned a few isolated words, such as father, mother, and potato, which she emits in a high-pitched voice. She has never been able to speak in sentences. Although crawling started at the age of 2 years, she could not walk unassisted until the age of 5 years. It was noted early in life that the child was mentally retarded. She had never been toilet trained and, up to the time of her hospital admission at the age of 17 years, she was wearing diapers. She has had measles at the age of 7 years, and at the age of 12 years, she had been seriously ill with an undiagnosed ailment, during which she ate or drank nothing for several days. Following this episode, she returned to her previous state of health.

At the time she was first seen in this institution (when 17 years old) she was dwarfish, measured 3 feet, 11 inches (=119 cm.) in height and had a weight of 36 pounds.



FIG. 2. L. M. Extreme wrinkling of the forehead, the beaked nose and the recessed chin are clearly seen here.

(=16.2 kg.) She walked with a stumping gait. She had a typical senile appearance, with wrinkled skin, thin panniculus adiposus, beaked nose, receding chin, and the hair of the capillitium was short, black, coarse, and had an average length of 4 inches and showed areas in which only a few hairs were present. (Fig. 1, 2) The pubic hair was scanty and that in the axillae almost absent. No hairs were seen otherwise on the body. The fingers were short and stubby. (Fig. 3) Hands almost identical with those of the present case are seen in the paper of Rene Broc et al who reported on typical infantile progeria. The toes were short, with the exception of the big toe and there was a wide gap between the big and the second toe. Particularly short was the third toe, compared with the second and the fourth. The toenails were defective, particularly on the right big toe where the nail was thick, gray, and



FIG. 3. The hands are small and the distal phalanges are extremely short, the nails are wider than they are long.

corrugated. The face had a peculiar bird-like appearance with a prominent hooked nose and a sharp mandibular angle which projects 3/4" over the anterior margin of the mandible. The skin of the face was wrinkled, and that of the trunk and extremities, except hands, smooth and somewhat loose. On the hands and the abdomen, the skin was markedly loose and showed numerous folds as in an old woman. There were vitiligo-like areas on the lower extremities and over the sternum. A white scar was present in the region of the right zygoma, measuring 1½" x 1" (in 1958). Another scar, 2½" x 1" was seen in the region of the proximal end of the right humerus. The teeth were crooked, overriding, and in a very poor condition. In 1939, at the age of 19 years, she menstruated for the first time. The menstrual flow was described as not copious and since that time she has continued to menstruate in minimal amounts and in a rather irregular fashion In 1955 she was treated for pulmonary tuberculosis for a period of 2 years, after which time she was returned to normal hospital life. She now measured 4 feet, 1 inch, (=124.5 cm.). This indicates a gain in body length of 5.5 cm. after the age of 17 years. Although the patient is a dwarf, there was a delayed cessation of growth. Her weight was, at that time, 52½ pounds (=23.8 kg.). Her heart showed a normal sinus rhythm, and her blood pressure was 112/76. Her blood cholesterol was 465 mg. per 100 ml. and the cholesterol esters were 300 mg. per 100 ml. The clinical examination of the sensory and motor systems was essentially within normal limits. The Romberg sign and the Babinski were negative. The ocular movements were normal. Her measurements in 1958 were as follows: spina iliaca anterior superior to malleolus internus, 28 inches (=70 cm.); spina iliaca anterior superior to lower margin of patella, 17 inches (=42.5 cm.); circumference of chest, 24 inches (=60 cm.); acromion to tip of middle finger, 21½ inches (=53 3/4 cm.); acromion to olecranon, 11½ inches (=18 3/4 cm.); circumference of head, 17 inches (=42.5 cm.) The distance from the incisura jugularis to the symphysis pubis was 16½ inches, (=41.25 cm.) and there is a moderate pectus excavatum present. The distal phalanges showed palmar flexion in all fingers. The distal phalanges were broad and the nails of all fingers centrally excavated. Although there was some prominence of the bones at the interphalangeal joints, no grossly discernible arthritic changes were seen. The musculature was fairly well-developed. The breasts were small and particularly the left smaller than the right. Cervical lymph nodes were palpable on the right side, questionable on the left. The thyroid was normal in size and consistency. The mouth was now edentulous. The buccal mucosa was normal and glistering. The palate was normal. The blood pressure was 90/60. At the time of admission, her I.Q. was 12, and the mental age, 2 years.

In June, 1958, a tumour was first noted in the right temporal region. It was palpable under the skin and was adherent to the subjacent structures. The tumour was surgically excised. It had the structure of a low malignancy fibrosarcoma (Fig. 4). Up to the present time, the tumour has not recurred, nor are secondaries present.

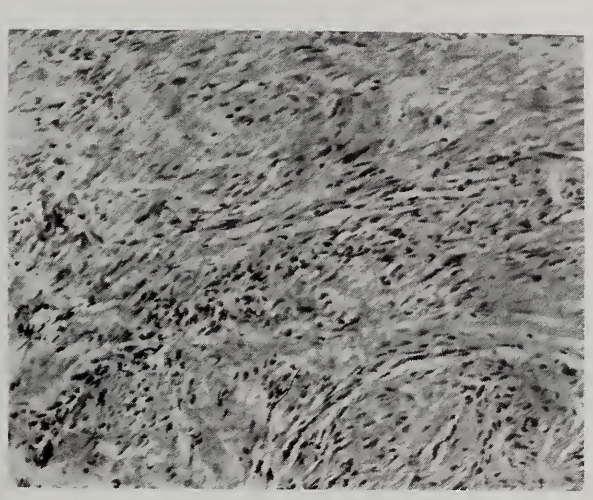


FIG. 4. Fibrosarcoma of temporal region. H. E. x 125.

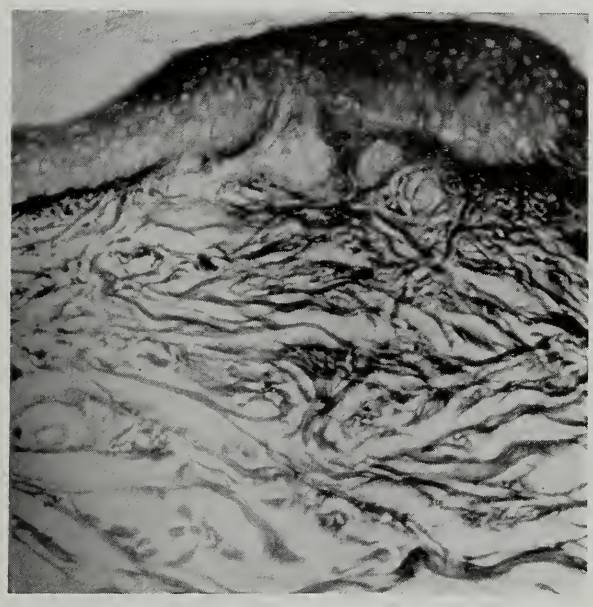
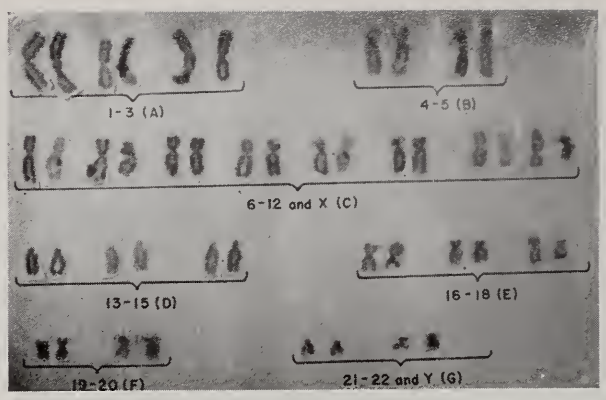


FIG. 5. L. M. Dermis Verhoeff van Gieson x 120.

An ophthalmological examination on March 30, 1964 revealed evidence of iridectomy on the left eye as the only pathological finding. It could not be ascertained when and why iridectomy was performed.

LABORATORY INVESTIGATIONS

The basal metabolic rate in 1958 was +37. A scraping of her buccal mucosa was sex chromatin positive. An electrocardiographic tracing done at this time showed a rate of 112 and no abnormalities. A skin biopsy was taken and the following stains were employed: haematoxylin-eosin, elastica-van Gieson, Heidenhain's azan, phosphotungstic acid haematoxylin, Gormori's aldehyde fuchsin. The findings were as follows: the epidermis is atrophic with almost absent epidermal pegs. In the cutis vera there are numerous melanophores and a few foci of cellular infiltration with histiocytes and an occasional mast cell. Sebaceous glands and sweat glands are present in the cutis vera, the latter mainly at the border with the subcutaneous fat tissue. An elastica-van Gieson stain shows a thin, normal, subepidermal elastic layer and in the deeper parts of the dermis,



Karyotype

numerous, coarse, curled, and partly fragmented fibres which take the elastic stain. (Fig. 5) In Heidenhain's azan stain, a few collageneous fibres show a red or bluish-red stain. With phosphotungstic acid haematoxylin, all fibres of the cutis vera which were positive with Verhoeff's stain were unstained, appearing pale yellow. After 10 minutes' treatment of a section with a 1% solution of trypsin at 37°C and subsequent staining with Verhoeff-van Gieson, only the elastic tissue of arteries and very scanty fibres of the dermis were elastica-positive, all other fibres of the cutis vera were red. With Gomori's aldehyde-fuchsin, the fibres were pale violet. All these findings indicate an elastoid degeneration of collagen (Lever[60]), (Ledoux-Corbusier[58]) and correspond to the findings in elastosis senilis (geroderma).

Her haemogram was normal. The I^{131} uptake was 44% in 24 hours. The Protein Bound Iodine by the Peters method was 5.5 mcg per 100 ml. (Normal range: 4-8 mcg) The Butanol Extractable Iodine (by the Peters method) was 4.0 mcg. per 100 ml. (Normal range: 3.2-6.2 mcg. per 100 ml.) Butanol Extractable Iodine was, therefore, 72.7% of Protein Bound Iodine, while the normal figure is 85% or more and PBI-BEI was 1.5 mcg. while this difference is normally no more than 1 mcg. The Total Iodine by the Barker method was 5.8 mcg. per 100 ml. which is within the normal range. In 1962, the Butanol Extractable Iodine was 4.0 mcg. per 100 ml. and 73% of Protein Bound Iodine. The Total Iodine was, at this time, 5.8 mcg. per 100 ml. BMR was repeated, and was at this time -21%. Cholesterol and cholesterol esters were also repeated and were 280 and 200 mg. per 100 ml. respectively. The Thorne test was normal and the α-aminonitrogen, by formol titration using the method of Rubenstein and Pryce was, on four occasions, 1.5 per 100 ml. Creatinine and creatine excretion in the urine were determined on six occasions. The figures were as follows: 1. Creatinine, 0.740 g; creatine, 0.078 g in 24 hours; 2. Creatinine, 0660 g; creatine 0.380 g in 24 hours; 3. Creatinine, 0.440 g; creatine 0.059 g in 24 hours; 4. Creatinine, 0.418 g; creatine, 0.045 g in 24 hours; 5. Creatinine, 0.385 g; creatine, 0.066 g in 24 hours; 6. Creatinine, 0.352 g; creatine, 0.079 g in 24 hours.

17-ketosteroids were, on April 5, 1963, 3.7 mg. in 24 hours and the ketogenic steroids, determined by the Norymberski bismutate method, 1.06 mg. After injection of 30 U. of ACTH, the 17-ketosteroids were 3.19 mg. on the first day and 5.65 mg. in 24 hours on the second day. The ketogenic steroids rose to 3.85 mg. on the first day and 5.39 mg. on the second day.

In another experiment, we administered 400 mg. of compound S (=17-hydroxy-11 desoxycorticosterone) over a period of 24 hours and evaluated 17-ketosteroids and ketogenic steroids prior to, during, and after the administration of compound S. The 17-ketosteroids were 2.73 mg. in 24 hours. With administration of compound S, they rose on the first day to 3.0 mg. per 24 hours and on the second day to 15.75 mg. in 24 hours. The ketogenic steroids were 2.80 mg. in 24 hours and with administration of compound S, they rose on the first day to 6.6 and on the second day to 9.98 mg. in 24 hours.

Metaphases of peripheral blood cells were prepared by a slightly modified method of Moorhead et al,[69] using the Difco TC chromosome culture kit and pretreating the incubated specimen with 1% trypsin solution. Ten metaphase nuclei showed all 46 chromosomes and a normal karyotype. The tables 1-3 summarize the symptomatology of progeria Hutchinson-Gilford, of Werner's syndrome, and of the present case.

TABLE 1

Symptomatology of Progeria	
Based on all (39) reported cases	
Males	60%
Females	40%
Not mentioned in one case	
Underweight	100%
Weight not reported on 3 cases	
Normal mentality	87%
Low mentality	13%
Mentality not reported in 9 cases	
Abnormalities of capillitium	100%
Not mentioned in 2 cases	
Short stature	100%
Prominent scalp veins	100%
Not mentioned in 4 cases	
Receding chin	100%
Chin not mentioned in 8 cases	
Abnormal teeth	100%
Teeth not mentioned in 3 cases	
Skin abnormalities	100%
Not mentioned in 4 cases	
Abnormal nails	100%
Not mentioned in 17 cases	
Skeletal abnormalities	100%
Not mentioned in 3 cases	
Beaked nose	100%
Not mentioned in 24 cases	
Full term delivery	67%
Premature	33%
Not mentioned in 12 cases	
Senility	100%
Not mentioned in 1 case	

TABLE 2

Symptomatology of Werner's Syndrome		
Based on 28 selected cases		
Males		54%
Females		46%
Cataracts present		93%
Cataracts absent		7%
Ulcers of skin		100%
Skin abnormalities other than ulcers		100%
Normal mental status		79%
Low mentality		3.5%
Mental status not mentioned		17.5%
Short stature		29%
Gracile extremities		43%
Stature not mentioned		43%
Normal stature		14%
Abnormalities of capillitium		96%
Normal capillitium		4%
Prominent scalp veins		
Receding chin		
Abnormal teeth	(Not mentioned in almost	
Abnormal nails	all reported cases)	
Skeletal abnormalities		
Beaked nose		

TABLE 3

Symptomatology of L. M.	
Underweight	+
Mentality	Low
Abnormalities of capillitium	+
Short stature	+
Prominent scalp veins	—
Receding chin	+
Abnormal teeth	+
Skin abnormalities	+
Abnormalities of nails	+
Skeletal abnormalities	+
Cataracts	—
Ulcerations of the skin	—
Gracile extremities	—
Beaked nose	+

Discussion

The tables summarizing the symptomatology of progeria, of Werner's syndrome, and of our case, show clearly the variability of symptoms associated with scleroderma-like cutaneous changes and cataracts on one hand, and the almost identical symptomatology of Hutchinson-Gilford's progeria and of the presented case on the other hand. The skin changes, although called usually scleroderma or scleroderma-like, are in the histologically examined cases not always the same (Strunz;[107] Knoth;[54] Matras and Kohler;[63] our case). However, in all cases, the essential change is a degeneration of collagenous fibres, which in our case assume the staining properties of elastoid and present the appearance of senile elastosis. These changes are the result of increasing "crosslinkage of protein spirals due to hydrogen bonding"[57] which occurs with aging. Although many cases described as Werner's syndrome show also some signs found in progeria and, as will be pointed out later, a pathogenic relationship is probable, it appears for the time being justified to separate typical cases of progeria in the adult from Werner's syndrome. Pathogenetic considerations of progeria were centered mainly on glands with internal secretion, particularly the pituitary. Short distal parts of extremities have been contrasted with acromegaly and considered as evidence of hypopituitaryism. However, the word acromicria is used for a symptom in different pathological conditions. Thus Benda[7] uses it for the hands of mongoloids and sometimes the "main en trident" of achondroplasia is called acromicria. Short hands are also present in Morquio's disease, in gargoylism, and in pseudohypoparathyroidism. The most detailed study of the skull in progeria is that of Sir Arthur Keith.[53] He compared the skull with that of "foetal ateleiosis" (Gilford). The latter name, literally "not arriving at perfection," indicates an arrest or delay which begins early in utero, while the delay in progeria is not uniform. Progeria is contrasted with acromegaly, ateleiosis with gigantism. However, eosinophilic adenoma of pituitary in childhood results in gigantism, not in typical acromegaly. The disparity in the development of cranial and of facial bones can be explained by the intrauterine time at which the delay begins, because the ossification of cranial, particularly occipital, bones preceeds that of facial bones (Arey.[2]) An abnormality of sella turcica was only exceptionally seen. (Exchaquet,[29,30] Zeder,[122,123] Thomson and Farfar.[112])

A few authors reported a low number of eosinophils using Rasmussen's counts. (Manschot;[62] Atkins;[3] Orrico & Strada.[79] However, it has to be borne in mind that the number of eosinophil cells in the pituitary is low in infancy and increases slowly, so that a general retardation of growth will necessarily result in a number of eosinophils lower than in a control of the same age. In addition, even laborious Rasmussen counts have a questionable value in view of changes which occur with alterations of nutritional state (Golden.[35])

As test for the activity of mucoid pituitary cells, their thyrotropic and corticotropic action was studied. The two estimations of basal metabolism showed such a discrepancy that no conclusions could be drawn (plus 37 and minus 21%). The former figure is probably erroneous because of the patient's restlessness. The uptake of radioiodide was 44% in 24 hours which, according to our experience, is normal (30-55%). The PBI and BEI were within the normal range, but the difference between the two, which normally is no more than 1 mcg. was, in this case, 1.5 mcg. The increase of this difference is common in juvenile goiter, but we found it also in several patients with mental retardation without goiter. The figures were not altered by injections of thyrotropic hormone. It indicates that a smaller fraction of PBI than normal is active thyroid hormone without the absolute amount of the latter being diminished. The cholesterol and cholesterol esters were raised on one occasion but normal on another one. Steinberg et al[105] reported "hypermetabolism" in progeria evidenced by increased uptake of radioiodide and aminoaciduria. However, their figures for iodide uptake are lower than our normals (30-55% in 24 hours) and the excretion of α-aminonitrogen was normal in our patient.

The figures for 17-ketosteroids and ketogenic steroids were low for the age, and corresponded to those of a young child. They agree with the few figures reported in the literature on progeria. The Thorn test was negative and the increase in the excretion of 17-keto - and ketogenic steroids after injection of ACTH was present but considerably lower than in normal adults. These findings do not indicate a specific involvement of pituitary or adrenals but a low functional level of both. The single case with anatomically demonstrable lesion in the adrenals (Variot and Pironneau[113]) is certainly an exceptional coincidence. After injection of 17-hydroxy 11-desoxycorticosterone (compound S) there was a marked increase in 17-ketosteroids and in ketogenic steroid indicating that there is no defect in the conversion of compound S in the liver into 17-ketosteroids, as assumed by Bauer and Conn[5] who found insufficient conversion. Our results are similar to those of Boyd and Grant.[12]

Another interesting finding is the marked creatinuria.

This is physiological in children and in old age and, in the latter, it can be abolished by testosterone. Creatinuria in our case may, therefore, be considered as result of arrested gonadal development.

The chromosomal count and the karyotype were normal female. The buccal mucosa was positive for Barr-bodies. This agrees with the finding of Moynahan[71] and of Djupesland[22] in two cases of infantile progeria. Considering the familial occurrence we may conclude that progeria is due to one or several pathological genes and not to a chromosomal abnormality. The endocrine glands, so far as they can be tested by clinical and laboratory methods, are all developmentally retarded and there is no reason to consider any one or all as primarily responsible for the disorder. An inherited enzymatic defect, as has been assumed, has no factual support. All previously discussed findings suggest that the essential pathology is a slowing down in the development of all organs and tissues which, at the same time, undergo the premature changes due to physiological wear and tear, characteristic otherwise for senescence (Shock.[103]) We suggest that progeria may be likened to familial heredodegenerative diseases of the Central Nervous System. The latter have been named by Gowers[36] abiotrophy and Raymond[87] considered them as "premature physiological senescene localized to certain organic systems (Raymond[87]) and ". . . the process is, in many cases, perhaps in most, an essential failure of vitality and we think it is instructive to consider degenerations in this aspect." (Gowers[36]) The presence of a single symptom in family members of progeria (Hutchinson's case, alopecia of our patient's father), and the occurrence of forms which begin in later age, are also familial and resemble only more or less infantile progeria or a patient like the one presented in this paper, who had all the symptoms of progeria except alopecia and arteriosclerosis suggest a multifactorial inheritance. We may consider our case as progeria surviving to middle age because of the absence of one manifestation, namely, atheromatosis. In patients in whom the disorder makes its appearance in later age, the clinical symptomatology becomes more variable and we have, on one hand, dwarfs resembling progeria, on the other hand, cases of Werner's syndrome where the only manifestations are skin changes and cataracts or "scleroderma" with infantilism without premature senescence (Guillain et al) and hereditary isolated geroderma (Bamater et al). All this suggests a mulifactorial genetic determination which, in the rare simultaneous presence of all pathological genes, produces true progeria of Hutchinson-Gilford; in the presence of a few of these genes results in the variable syndrome of Werner. Each of the concerned genes controls the maturation and resistance to physiological wear and tear of a tissue, organ-system or organ and the gene action may become manifest in foetal or in postnatal life.

An unusual symtom and sign in our case is mental retardation and microcephaly. This is, we believe, not a manifestation of progeria but the result of perinatal anoxaemia associated with the baby's immaturity. Although full term, the baby had a very low birth weight, indicating retardation of growth starting in foetal life, and predisposing the newborn to birth injuries (including perinatal anoxaemia) which are characteristic for the premature infant. The presumable diagnosis is, therefore, sclerotic atrophy of the brain and microcephaly resulting from perinatal anoxaemia.

Summary

The literature on infantile progeria and that on Werner's syndrome are reviewed. A case of progeria in an adult is described, and the symptomatology compared with that of Hutchinson-Gilford's progeria and that of Werner's syndrome. Endocrinological, metabolic studies and findings in peripheral blood-cell culture are reported. A hypothesis is put forward which considers progeria as abiotrophy of all tissues and organs and is determined by multifactorial inheritance. The individual gene's effect becomes manifest at various times of foetal or postnatal life. The genes determine the velocity of development and the onset of wear and tear changes. Simultaneous presence of all these pathological genes results in typical progeria, the presence of a few and later active genes leads to the variable syndrome of Werner.

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Function of the Clinical Psychologist in Private Practice

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The much-propagandized team approach in the mental hygiene field has shed some light on the functions of persons trained to deal with the multiple problems presented in a typical community mental health clinic. Ranging through psychological, medical, and social areas, the services of the team encompass diagnosis, medication, and psychotherapy. These services include family therapy, or counseling and environmental manipulation; in short, they frequently encompass the totality of life problems which people bring to those who attempt to alleviate distress.

The clinical psychologist, as a member of such a team, serves a number of functions in the many-pronged approach of today's community mental health clinics. He contributes to personality assessment, he is engaged in psychotherapy on any number of levels and within different contexts, and he may be engaged in research on any one of these areas which, in themselves, have proven to be thorny problems about which more systematic knowledge is required. Thus, the clinical psychologist as a team member is no longer an unknown quantity among those who have occasion to work with the resources of a community mental health clinic.

Frequently, however, the function of the clinical psychologist practicing in the community at large is not well known. It is the purpose of this paper to delineate several areas of concern, areas in which the clinical psychologist may contribute his share in the alleviation of personal distress within the framework of a given community in general and in collaboration with the medical community in particular.

In order to explicate the function of the clinical psychologist, it may be proper to start with a brief outline of his training and qualifications. Subsequent to the completion of the requirements of a B.S. or B.A. degree with a major in psychology or one of the sciences, graduate training usually involves a rigorous four-year program, during which time psychology as the science of behavior is studied in depth. Because a Ph.D. degree is an academic or "teaching" degree, the problems studied include perception, motivation, learning and forgetting, conditioning, and physiological substrata of behavior. For those who choose specialization in the clinical field, the clinical aspects of psychology in addition to the academic become a focus of study. Concern with problems of personality development, attempts at altering unhealthy behavior, research into differential methods of such therapeutic interventions, and techniques of psychometrics take on special significance.

The graduate student in psychology supplements academic work with practicum experiences under close supervision in university-affiliated clinics, hospitals and schools. At the termination of the primarily academic-and-practicum phase, which lasts three years, the student takes an internship, which frequently is served in a V.A. hospital or in a university-approved state hospital or clinic. Internships are approved in only those installations which facilitate the full range of functions of clinical psychology, such as individual and group psychotherapy, personality assessment, differential diagnosis, and research. Interns are required to present their diagnostic assessments and therapeutic work at departmental and full-staff conferences. Another important value of the student's intership in such a multi-disciplinary setting stems from his close proximity to and mutual experiences with members of other helping professions and his first-hand view of the ramifications of public-health aspects. To foster research, the student is required to design, execute, and write an original research project which may serve as one requirement for the Ph.D. degree. The psychologist usually enters into affiliation with the national organization, the American Psychological Association, as well as state and local psychological associations.

The Clinical Psychologist in the Community

We come now to the question of what services are being rendered by a clinical psychologist practicing in the community. As previously indicated, one area of concern is personality assessment and diagnosis; i.e., the delineation of a person's salient characteristics, the motives underlying his observable behavior, and the classification of his major characteristics within current nosology. In matters of diagnosis it should be emphasized that contemporary clinical psychology de-emphasizes neatness of labeling or elegance of categorization, but rather attempts to develop a set of terms which will aid in the clarification of understanding. The underlying assumption is that categorization tends toward sterile disposition, whereas a clear understanding tends to fruitful change or amelioration. Clinical psychology is also intimately involved in the wide area of behavior change, whether it be called psychotherapy or some other name. Formally or informally, the practitioner will usually go through a process of assessment and prior planning before undertaking or instrumenting attempts designed to modify behavior. In certain otherwise equivocal cases, when the clinical psychologist contributes his understanding of personality assessment, such planning can be clarified, a regimen worked out, certain difficulties may be anticipated, and there may

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be some prediction of possible outcome. The point to be made is that through interview and diagnostic tests the psychologist can tell the physician how sick his patient is under the presented symptoms, what the future course of the illness is likely to be, and what outstanding emotional conflicts are under the surface and causing the illness.

The Clinical Psychologist in Diagnosis

The work of the clinical psychologist in diagnosis may be exemplified by the three following cases, which are typical of those that frequently come to our attention. As indicated above, successful diagnosis involves questions revolving around the management of the case, the treatment plan, and possible further diagnostic study by other means if pertinent, i.e., EEG, audiometry, neurological evaluation, etc.

Case 1 is a sixteen-year-old high school student whose academic performance has dropped markedly and fairly suddenly over the past semester. His parents report that he "has lost all interest and stays at home all the time," and that he seems to have broken contact with his friends. They also report that it is difficult to get him to follow the usual personal hygiene routine and that he frequently refuses to change his clothes. On examination, no organic evidence which might explain the manifest behavior is elicited, and neither are there any overt manifestations of psychosis, such as bizarre mannerisms, delusions, and hallucinations. Question: Is this a case of "adolescent turmoil," i.e., a transient phase, perhaps here somewhat extreme but essentially benign; or is there a latent pathologic process at work, such as schizophrenia just beginning to emerge but not yet clinically overt? Psychological testing (including an assessment of current intellectual functioning) will attempt to measure the effect of family and environment on basic personality. This information is helpful in obtaining a decisive measure of understanding of the ongoing situation as a basis for a decision as to appropriate treatment.

Case 2. A fifty-eight-year-old business man, has complained for several months about defective memory, increased irritability, occasional diplopia, headaches, and occasional sensory numbness in both upper extremities. His family reports that he has "become different from what he was before." His history includes an automobile accident one year ago but without evidence of intracranial injury. Question: Are these symptoms sequelae to the accident and do they represent some organic brain damage of as yet unknown etiology? Do the symptoms perhaps suggest an intracranial neoplasm, which may be operable? Or, in the light of increasing negative neurological and other findings, are these symptoms masking a depression, or are they hysterical manifestations? Psychological testing, specific for organic deficit and assessment of the salient primary personality features, can be useful in contributing an answer to these questions.

Case 3 is nine years old, is obviously slow in schoolwork, reads very poorly, and plays with younger children. He has a bilateral congenital hearing impairment and wears a hearing aid. Question: What is patient's level of intellectual functioning? Are there areas of sensory disturbance other than auditory which contribute to the observed behavior? Are there other reasons for his school difficulties? Is he insufficiently motivated for school because of emotional problems? Is he able to perform better on some of the various aspects of intellectual functions than on others? How can this information be used to help the school to plan for him? Once again, psychological study offers practical information.

Testing should not be a "shot-gun" method that subjects the patient to an expensive, time-consuming technique which may easily arouse emotions of anxiety. Rather, psychological tests should be used selectively with specific questions in mind or with a working hypothesis for which confirmation is sought. The answers obtained from test data never stand alone but are considered within the entire clinical picture.

Development of Effective Psychotherapeutic Methods

Clinical psychologists are involved in the development of effective psychotherapeutic methods. Psychotherapy is another function of clinical psychologists, and following are three cases which represent a sample of those frequently seen in private practice.

Case 1. A seventeen-year-old attractive high school student from an intact, middle-class home, was referred following a suicidal gesture which had been preceded by periods of depression and generally turbulent behavior. Despite initial parental resistance, based on inability to understand their daughter's motivation, the youngster accepted a regimen of regularly scheduled sessions and began to "unload" some of her experienced, but heretofore not verbally expressed feelings. Feelings of inadequacy and much repressed anger, as well as the need for a model, were crucial aspects in the ensuing psychological treatment process. The family physician maintained a consulting role and provided the parents with emotional support. As the youngster became able to function more adequately, frequencies of interviews were reduced. After six months she was seen only occasionally.

Case 2. This patient was thirty-two years of age, married, an engineer with two children, who complains of abdominal pain, fatigue, headaches, and crisis states, for which emergency medical treatment was sought. The history of this condition was of two to three years' duration, involving numerous medical work-ups in clinics and private "doctor shopping," with a tentative diagnosis of duodenal ulcer. At the time of referral there was no evidence of any organic basis for the patient's experiences, which were rapidly threatening to become totally disabling. The patient's physician initiated referral with the request to assess the psychological problems, which appeared to be of primary importance in the patient's symptoms. Psychotherapy, with regularly scheduled, semiweekly sessions, enabled the patient to give up most of his symptoms after two months and then started him on working out his underlying psychological problems to such a degree that, when similar stress was placed on him as in the past, he did not use the same self-defeating defense mechanism, but was able to deal with the situation more directly. Initially, the referring physician maintained the patient on tranquilizing medication until the symptoms cleared and anxiety diminished to a degree where the patient could handle it.

Case 3. A forty-year-old housewife and mother of three children, suffered from insomnia and what appeared to be a deepening depression. She cried easily, sometimes for much of the day, had difficulty getting up in the morning, complained to the physician that she felt "dragged out" and exhausted, was unable to do her work, and lost interest in the activities she had previously been engaged in. Her appetite had fallen off and she had lost weight. She responded momentarily to efforts by her minister to talk with her and to draw her back into activities, and she responded to efforts of her physician to talk with her when he was able to do so. However, her response did not last. Psychiatric consultation confirmed the diagnosis of Reactive Depression. A regimen of appropriate anti-depressant medication was coupled with referral to the clinical psychologist for regularly scheduled psycho-

therapeutic sessions, during which the patient eventually was able to ventilate her suppressed feelings and, later, to restructure her internal attitudes.

The question is often asked: "Are there more emotionally disturbed people today than there were previously, or are people going along with a fad in seeking out psychiatric or psychological aid for problems?" Undoubtedly, new and different stresses on people in contemporary social settings result at least partially in new and different strains within people. Each individual has to find his own ways and means to cope with such strains, but individuals often adopt inefficient and eventually self-defeating means, with the result that more stress is created. The same inefficient or self-defeating mechanism is then used again, thus creating a vicious circle. It is frequently impossible for an individual to break out of this established pattern of behavior without skilled aid.

The psychologist has to seek out and bring to light the elements of motivation, feelings, and other "unpleasant" psychological manifestations which a person often hides successfully from himself. With his special knowledge and training, the psychologist can be of great aid to the physician in dealing directly with underlying psychological problems, as well as offering diagnostic services.

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The Doctor as Musician

EDWARD PODOLSKY, M.D.

Apollo was the god both of medicine and of music. The priest-doctors of ancient Egypt and the medicine-men of the American Indian tribes were also musicians. They used both music and medicine to heal the sick of mind and body.

All the ancient peoples knew of the healing power of music. They had a musical healing treasury of great worth. Now, after several thousand years, the names of Sarpander, Arion and Zenoceates are still known as men who made good use of music in healing disease.

There has always been a sympathetic relationship between music and medicine and between physicians and musicians. Many medical men have contributed as much to the development of music as to the development of medicine. Among the earliest of noted English composers was George Ethridge who lived during the 16th century and was one of the most famous vocal and instrumental musicians of his day. He was a graduate of Oxford and a physician of great ability. Towards the end of the 16th century Sir Thomas Gresham established a professorship of music at Oxford. Curiously enough, the first five men to hold this chair were all physicians. They were masters of both arts.

Among the earliest musical compositions extant by medical men are those of Thomas Campion, who was born in London, February 12, 1567. His early interests lie in medicine and he took his M.D. at Cambridge. Following his graduation he took part in Lord Essex's expedition which landed at Dieppe in 1591 and laid seige to Rouen. As a physician he gained admission to the London Tower to visit his friend Sir Thomas Manson who was accused in the complicity of the murder of Sir Thomas Overbury.

After his military adventures Campion became very much interested in music. The first musical composition was "A Book of Ayres Set Forth To Be Sung To the Lute Orpheriam and Base Violl." This appeared in 1601. Three more books of airs followed within the next sixteen years.

Dr. Campion also wrote several masques, both words and music, for special occasions. Among these was a masque performed at Whitehall on Twelfth Night, 1607, in honor of Sir James Hay. Another masque was performed in 1613 at the Banqueting House in Whitehall at the marriage of Frederick Balantine with the Princess Elizabeth, for one song of which he wrote the music. In the same year he wrote a masque for an entertainment in honor of Queen Ann, wife of James I, and another masque by him was performed on the occasion of the marriage of the Earl of Sommerset and Lady Francis Howard.

Dr. Campion was also a musical theorist of note; his "New Way of Making Foure Parts in Counterpart by a Most Familiar and Infallible Rule," published shortly before his death, went through many editions. He died on March 1, 1616, and was buried at St. Dunstan's.

Probably the most famous of the early English doctor-musicians was Henry Harington. His "Great is the Pleasure" is one of the most popular of musical compositions and it has been played and sung in all quarters of the globe. He was born in Kelston, Somerset, England, on September 29, 1727. In 1745 he entered Queen's College, Oxford, with the intention of taking holy orders. He used to pass his cavarions with his uncle, William William, vicar of Kingston Wilts, from whom he inherited a taste for music. In 1748 he took his A.B., and shortly thereafter gave up his intentions of taking holy orders.

Harington thought that medicine offered a more attractive career and for this reason he remained at Oxford taking his M.A. and later his M.D. But his love for music was already manifested in many ways. While at Oxford he joined an amateur musical society established by Dr. W. Hayes to which only were admitted those who were able to play and sing.

When he left Oxford, Harington entered medical practice at Bath. All his leisure time was devoted to music and composition. He was in time appointed "composer and physician" to the Harmonic Society of Bath of its founding in 1784 by Sir John Davies.

Two books in folio of Dr. Harington's glees were issued in 1785. Later other glees followed. In 1800 he published "Eloi! Eloi! Or the Death of Christ," a sacred dirge for passion week.

Harington was also much interested in civic affairs. He was the first alderman of Bath and later Mayor of that city. His compositions were distinguished for originality, correct harmony and tenderness.

Another famous doctor-composer of the 18th century was William Kitchner. He was born in London in 1775, the son of a coal merchant from whom he inherited a comfortable fortune. He was educated at Eton and Glasgow where he received his M.D. But his interest lay mainly in music. He composed an operetta "Love Among the Roses." He was also the author of a musical drama, "Ivanhoe."

Dr. Kitchner also made notable contributions to musicology, being the author of "Observations on Vocal Music," and editor of "Loyal and National Songs of England," "The Sea Songs of England," and "A Collection of Vocal Music in Shakespeare's Plays."

Literature was another field in which Dr. Kitchner distinguished himself. He was the author of some rather unusual literary works, among them, "The Cook's Oracle," "The Art of Invigorating and Prolonging

Life," "The Housekeeper's Ledger," "The Economy of the Eyes," and "The Traveler's Oracle." His medical views were rather eccentric, but his music was wholesome and pleasing.

By no means were the doctor-musicians all English. Florient Cornielle Kist was among the most famous of Dutch musicians. He was born in Arnheim, Holland, on January 28, 1796. He took his M.D. at the University of Leyden and from 1818 to 1825 he practiced medicine at the Hague. He was a flutist and cornetist of great ability, and among the greatest compositions written for these two musical instruments are to be found many which were composed by Kist.

Dr. Kist was a founder of the Diligentis Society at the Hague and later of the Caechilia which is still the most important musical society in Holland. He was also the founder of the Choral Union and Collegius Musicum at Delft.

In 1814 Dr. Kist settled at Utrecht where he became the director of the Netherlands Musical Times which he edited for more than three years. Dr. Kist wrote many cantatas and vocal compositions which were extremely popular in their day. His influence on Dutch music was profound. He was one of the most important of all Dutch musicians and composers.

Perhaps the greatest of all the doctor-musicians were Louis Hector Berlioz and Alexander Porfyrevich Borodin. Berlioz's father, a general practitioner in a village on the outskirts of Lyons, France, had always hoped that his oldest son, Hector, would follow in his footsteps. However, Hector's interest and great love for music had manifested itself early in life. As a matter of fact his father was also a musician and when Hector was twelve years old his father taught him to read music. Within six months the young musician had made such progress that he had composed a potpourri of Italian airs, as well as two musical quintets.

Because of the constant insistence of his father, Hector studied medicine in which he had little interest; he was interested only in music. In 1823 Hector Berlioz, a musician at heart, became a doctor. He practiced medicine for three years and in 1826 entered the conservatory of music to round out his musical education. Four years later he composed his famous "Symphonie Fantastique."

Thereafter, Berlioz devoted himself exclusively to music. Among his more outstanding works are his "La Damnation de Faust," a dramatic legend in four parts, considered his greatest symphonic work after "Symphonie Fantastique," "Episode de la Vie D'Un Artiste," "Romeo et Juliette," dramatic symphony: "L'Enfance Du Christ," a trilogy; "Harold en Italie," a symphony in four parts: the overture to "King Lear"; and the operas "Les Troyens a Cartharge," "Benvenuto Cellini," and "Beatrice et Benedict."

Alexander Porfyrevich Borodin was the natural son of a Russian prince and was born in St. Peterburg on November 12, 1834. He was educated in medicine and in 1862 was appointed assistant professor of chemistry at the St. Petersburg Academy of Medicine. He was the author of several books on chemistry which attained a great deal of popularity.

Dr. Borodin was also very much interested in medical education and he took a leading part in advocating medical education for women. He helped to found the school of medicine for women and he lectured there from 1872 until his death.

However, it is as a musician that Dr. Borodin is best known. His interest in music was stimulated in 1862 by his friendship with the great Russian musician, Balakirev. His wife was also very much interested in music and she helped him keep this interest alive in her husband. Dr. Borodin was encouraged greatly by Liszt with whom he kept up a long correspondence.

Borodin's first symphony was written in 1862-67 and this won favorable notice at once. His greatest musical composition was the opera, "Prince Igor," which he began in 1869 and left unfinished at his death. It was completed by Rimsky-Korsakov and Glazunov in 1889. This attained great popularity in Russia with its brilliancy Polovstienne dances became famous throughout Europe as a consequence of the performance by the Diaghilev Ballet.

In the field of music Borodin's symphonic sketch, "In the Steppes of Central Asia," is well-known. His second symphony in B minor is of the first rank. He also wrote part of a third symphony, a couple of quartets and many delightful songs.

Dr. Borodin's total musical output is not very large, but it represents among the greatest musical work of all time. He is at this date one of the most popular of Russian composers, and he is heard as frequently as are Rimsky-Korsakov and Tschaikovsky.

Alexander Borodin died in 1887 at the early age of 53. Had he lived longer there is no doubt that he would have taken his place among the greatest composers of all time. He remains one of the two greatest doctor-composers, a credit to his two great professions.

The list of doctor-musicians is, unfortunately, a rather small one, but also a very select one. The individual contributions of each man are of the highest order and of the greatest importance. Doctors have always been great lovers of music. In Vienna many years ago the Doctors' Orchestra was very popular. Today the New York Doctors' Orchestra gives concerts which are well attended and greatly appreciated. In other large cities, doctors are forming musical aggregations to give vent to their musical tastes.

1049 East 18th Street, Brooklyn 30, New York

INTERIM MEETING

Maine Medical Association House of Delegates

Sunday, April 11, 1965

THE STOWE HOUSE, BRUNSWICK, MAINE

Dinner at 1:00 P.M. followed by the Business Meeting
Presiding, Linus J. Stitham, M.D., Dover-Foxcroft
Speaker of the House of Delegates

The Order of Business will include financial statement for 1964, proposed budget for 1966 and other matters to be acted upon at the Annual Meeting in June.

Complete agenda for this meeting will be mailed to each of the county delegates and alternates.

Maine Medical Association Council

The Council will meet at the M.M.A. headquarters in Brunswick, Maine at 10:00 A.M.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

A Problem in Toxicology

E. W. CAMPBELL, DR.P.H.*

Probably the outstanding problem encountered in the administration of the toxicology laboratory is the lack of adequate amounts of materials sent in for desired analyses. In a nutshell, that is the story. It is a problem, however, which is not immediately discernible through the current available literature on the subject. Thus it seems pertinent to bring this to the attention of those physicians, or others, who occasionally have need to send in materials for analysis and, of course, wish and deserve the best possible results from the materials submitted. To illustrate:

Early one morning a young couple were found dead in a parked car. The bodies had the typical coloration for carbon monoxide poisoning, or seemingly so, and thus a tentative diagnosis of death from this cause was made. However, an autopsy was ordered and a small amount of material taken for analysis – approximately 5 cc. of blood – and, because of the circumstances, a request was made for carbon monoxide testing only. Without further activity and before the analysis could be obtained, the bodies were released to an undertaker. When the serological material arrived at the laboratory, analysis for carbon monoxide showed a low percentage in one body – less than 30% saturation – and an even lower percentage in the other, or a matter of 10% saturation. Obviously, other determinations were necessary, but the material sent in had all been used in making the prior determination. A request for more material was made. Unfortunately, the bodies had been embalmed and those determinations which should have been made immediately – such as testing for methyl alcohol and many other compounds – were no longer possible.

With great difficulty additional material was secured. The upshot of the matter was that one victim was found to have ingested a mixture of barbiturates; the other, a large amount of alcohol. How simple it would have been at the time of the autopsies to have collected some 4-6 ounces of blood, possibly some of the organs or portions of these, on which to make other important toxicological determinations!

*Director, Division of Sanitary Engineering.

Accidents can happen in laboratories, too. For example: if serological samples of only 2-3 cc. for blood alcohol determination are submitted and an accident of spillage occurs, it is practically impossible to obtain an adequate result. And even though it is theoretically true that a determination can be made on as small an amount as 1 cc. the normal routine is to duplicate this amount which generally uses up all of that material. Thus, if further determinations are requested, such as testing for barbiturates, it is obviously impossible to do this under the circumstances. In many cases of suspected drunkenness, there is also the possibility of drug ingestion, and in numerous cases, a possibility of carbon monoxide – perhaps, a combination of all of these.

For legal purposes, it is perhaps sufficient to make analysis only for alcohol content but in all fairness to the victim, analysis should also be made for a variety of other substances. Plainly, these cannot be accomplished with a sample of only 5 cc.

In the case of autopsy materials, there is usually no problem. Each medical examiner or pathologist should arm himself in advance with a few jars, 8-ounce bottles or similar containers for liquids, as well as suitable containers for portions or organs that need to be packed separately. It is very important that all organs be kept separate from each other. Sometimes plastic bags will suffice for transportation purposes so long as organs are well separated. It scarcely needs repeating that samples should be kept under refrigeration whenever possible and transported promptly to the laboratory for the desired determinations.

It is important, too, that no anticoagulants, preservatives, deodorants or any other substances be added to the specimens if chemical or toxicological analyses are desired. If such materials are added before the need for investigation has been determined, then complete information should be furnished the laboratory as to the kind of materials added, as well as submitting a portion of the added product for comparison and analyses.

In summary, it cannot be stated strongly enough that if the toxicologist is to do justice to the materials submitted for analyses, a sufficient quantity of liquids must

be sent. With autopsy materials, half or even whole organs should be submitted in the manner indicated above, for the rest of the analyses.

The following table is offered as a guide to those who may wish to send samples to the State Toxicological Laboratory for analysis:

TABLE I

MINIMUM AND DESIRED QUANTITIES OF TOXICOLOGY SAMPLES NEEDED FOR ANALYSIS

	Minimum	*Desired*
Alcohol (Ethyl)		
Blood, brain tissue, gastric content, or urine	5 ml. or 5 grams	10 ml. or 10 grams
Wood Alcohol (Methyl)		
Blood, brain tissue, gastric content, or urine	5 ml. or 5 grams	10 ml. or 10 grams
Barbiturates		
(Qualitative and quantitative but without specific identification of barbiturate)		
All samples (blood, brain tissue, gastric content, and urine)	10 ml. or 10 grams	20 ml. or 20 grams
(Determination and identification)		
All samples	50-250 ml. or grams (depending on concentration)	250-500 ml. or grams
Alkaloids (including both plant and synthetic organic bases)		
All samples	100 ml. or 100 grams	250-500 grams
Carbon Monoxide		
Blood	5 ml.	10 ml.
Methemoglobin		
Blood	5 ml.	10 ml.
Porphrins		
Urine	25 ml.	50 ml.
Arsenic		
Blood, gastric content, urine, liver tissue or kidney tissue	10 ml. or grams	50 ml. or grams
Hair or finger nails	1 gram	10 grams or more
Mercury		
Blood, gastric content, urine, liver tissue, or kidney tissue	50 ml. or grams	200 ml. or grams
Lead		
Blood	10 ml.	50 ml.
Urine	50 ml.	200 ml.
Other Heavy Metals		
Blood, gastric content, urine, liver tissue or kidney tissue	100 ml. or grams	300 ml. or grams
Cyanide		
Gastric content	5 ml. to 100 ml. (depending on concentration)	200 ml. or grams
Other samples	100 ml. or 100 grams	200 ml. or grams
Fluoride		
Gastric content	25 ml. to 100 ml.	100 ml.
Other samples	100 ml. or grams	200 ml. or grams
Phosporus (white)		
Gastric content	25 ml. to 100 ml.	100 ml.
Other tissue	200 ml. or grams	400 ml. or grams
Benzene		
All samples	50 ml. or grams	200 ml. or grams



AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Lung Cancer Detection

A simple and rapid method of detecting and locating lung cancers has been devised by scientists at Johns Hopkins University School of Medicine. Lung cancer is only four or five per cent curable because it is so hard to detect in an early stage.

While its actual percentage performance is yet to be determined in untreated patients with early and otherwise undiagnosable disease, the results so far indicate that the technique will be of great value in identifying some lung cancer patients among patients with chest ailments.

This was reported recently by the American Cancer Society which supported early development of the technique by Dr. Curt P. Richter many years ago. The U.S. Public Health Service has financed tests more recently. Dr. James F. Fries worked with Dr. Richter in studying lung cancer patients.

Twenty years ago Dr. Richter began making "electrical skin resistance tests" on patients with a variety of diagnosed and undiagnosed diseases. Over the years, in thousands of such tests he found the procedure of value in locating internal tumors, sources of referred pain, diseased segments of nerves, blood vessel abnormalities and other troubles.

The technique consists essentially of sending a light battery current through various pathways of the patient's body. The current is so slight that it can scarcely be felt; in the thousands of tests it has proved non-traumatic to every patient. The current is conducted by tissues and fluids under the skin as efficiently as by metal wires. All resistance to the current normally is centered in the skin itself.

The sympathetic nervous system, which automatically regulates the response of skin to stimuli, controls the skin's resistance to the current; the more active the sympathetic nerve, the less resistance the current will encounter. If a sympathetic nerve is severed, resistance to the current along this pathway will be enormous; if it is compressed or damaged (as by a tumor) the condition will register in increased resistance to the current.

The equipment and test devised by Dr. Richter are extremely simple. Essentially, the equipment is composed of two small batteries (22½ and 90 volts), wires, two electrodes and meters which record the flow of current and resistance to it. It is portable, inexpensive and operated by a technician.

One electrode is attached to the patient's leg; the other — a small metal wheel — is run methodically over specific skin areas. The electrical resistance of various skin sites is recorded by meters and mapped by the investigator on a drawing of the human body. A hot room (at 100 degrees, Fahrenheit) enhances the system's sensitivity.

A technician can run an electrical skin resistance test for lung cancer in 10 minutes.

Drs. Richter and Fries have found that lung cancer patients yield four kinds of resistance patterns — all of them different from the normal. The telltale resistance set up by lung cancer registers in various skin areas of the head, neck, shoulders, chest and arms. Various patterns represent the location of the tumor and its colonies with respect to specific sympathetic nerves. They also reflect tissue damage from prior surgery or radiation.

In 37 patients with proved lung cancer, all tests were positive. In 35 patients with assorted other diagnosed and undiagnosed chest conditions (including tuberculosis, syphilis, emphysema, sarcoidosis and others) and matches with the lung cancer patients for age, cigarette smoking history and some symptoms, only one showed a lung cancer pattern.

The technique discloses the existence and location of at least some metastases, or cancer colonies, and this has proved of clinical value. Patients with cancer too disseminated for effective treatment can be saved the trouble and expense of radical surgery in a vain attempt to cure.

The investigators believe the test probably will show up all early cancers which affect sympathetic nerves. They do not know what percentage of lung cancers might be detected in a curable stage, because they do not know what proportion of them press upon or damage nerves before they spread to other tissues. Further tests eventually will establish the true value of the technique.

Release from American Cancer Society News Service



The Doctor's Visit, Jan Steen 1626-1679, Mauritshuis, The Hague

In Diverticulosis and Diverticulitis...

METAMUCIL® brand of psyllium hydrophilic mucilloid

"Diverticulosis ... a low-roughage diet is advisable.... Constipation is avoided, preferably by the daily use of Metamucil.

"Diverticulitis Mild, chronic symptoms of diverticulitis, such as diarrhea or flatulence also are treated[1] by low-roughage diet, adequate fluid intake and Metamucil...."

Usual Adult Dosage: One rounded teaspoonful of Metamucil (or one packet of Instant Mix Metamucil) in a glass of cool liquid one to three times daily.

Metamucil is available as Metamucil powder in containers of 4, 8 and 16 ounces and as flavored Instant Mix Metamucil in cartons containing 16 and 30 single-dose packets.

1. Welch, C. E., Diverticula of the Alimentary Tract, in Conn, H. (editor): Current Therapy—1961, Philadelphia, W. B. Saunders Company, 1961, pp. 224-225.

SEARLE

Research in the Service of Medicine

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County Society Notes

ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held on December 17, 1964 at the Central Maine General Hospital in Lewiston, Maine. The President, Robert D. Wakefield, M.D. called the meeting to order.

Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association, explained the procedure by which the payment to the Maine Medical Education Foundation was changed from a voluntary contribution to an assessment and it was voted to approve this change.

Rudolph Haas, M.D. proposed that the society go on record as supporting Dr. Fisher's supplemented budget, in particular the increase of visiting public health nurses and funds for the diagnostic laboratory which was approved.

The annual meeting of the Corporators of the Androscoggin County Medical Association was held at the Central Maine General Hospital with twenty-eight members present.

The treasurer's report was read and Otis B. Tibbetts, M.D. gave a report for the financial committee which were both accepted.

Lawrence A. Nadeau, M.D. presented the report of the nominating committee and the following slate of officers were elected for 1965:

President, Henry C. Thacher, M.D., Auburn
Vice-President, Harvey J. Proulx, M.D., Lewiston
Secretary-Treasurer, Donald L. Anderson, M.D., Lewiston
Delegates to the Maine Medical Association House of Delegates: Drs. Robert D. Wakefield (1 yr.) and Morris E. Goldman (3 yrs.), both of Lewiston. Alternates: Drs. Henry C. Thacher (1 yr.) and Charles A. Hannigan (3 yrs.), Auburn
Councilor, Louis N. Fishman, M.D., (3 yrs.), Lewiston

Dr. Wakefield was given a rising vote of thanks for his efficient year's service.

The Androscoggin County Medical Association met at the St. Mary's General Hospital in Lewiston, Maine on January 21, 1965 with twenty-two members present. The meeting was called to order by the President, Henry C. Thacher, M.D.

Dr. Thacher appointed a committee consisting of Drs. Michael J. Harkins, Charles W. Steele and Paul M. Beegel to inquire about the Vocational Rehabilitation Department status.

The guest speaker, Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, was introduced by Daniel R. Shields, M.D. The development of Blue Shield fees was explained. At present our fees are based on the 1956 California relative value scale which was more exact than the old trial and error method. A new fee schedule is being formulated based on the 1960 revision. A lively question and answer period followed.

DONALD L. ANDERSON, M.D.
Secretary

CUMBERLAND

A meeting of the Cumberland County Medical Association was held in the ballroom of the Eastland Motor Hotel in Portland, Maine on January 21, 1965. The members of the Woman's Auxiliary to the Cumberland County Society were invited to this meeting and there was a total attendance of 196.

Following a social hour and dinner, the President, Alvin A. Morrison, M.D., accepted a motion for omission of any society business so that George F. Sager, M.D. could introduce the speaker of the evening, Mr. Steve Merrill.

Continued on page 64

Maine Heart Association Notes

Diuretics and Electrolyte Abnormalities in Congestive Heart Failure

"The retention of sodium and water which characterizes the renal response to heart failure is an important factor in the development of hypervolemia and is responsible for many of the subsequent manifestations which appear. Logically, therefore, one of the major goals of therapy is to increase sodium and water excretion and prevent their subsequent retention. The use of diuretics in the treatment of congestive failure has increased steadily since . . . recognition that diuresis could be produced by the administration of mercurials. Indeed, diuretic administration now constitutes the most important aspect of any good therapeutic regimen in the long-term treatment of this disease. Unfortunately, diuretics may also be responsible for a number of complications that commonly occur."

Reference: Walker, W. G. and Cooke, C. R. Modern Concepts of Cardiovascular Disease, Volume 34, pages 7-11, 1965.

COUNTY SOCIETY NOTES

Continued from page 62

Mr. Merrill told of his one year in medical school, his background leading to his being "funny for money," and of his concept of Maine yarn-spinning, including many tall tales of up-state Maine folks.

STANLEY B. SYLVESTER, M.D.
Secretary

KENNEBEC

The Kennebec County Medical Association met on January 21, 1965 at The Senator in Augusta, Maine. Following dinner, the President, John D. Denison, M.D. called the meeting to order.

Richard H. Dennis, M.D., Chairman of Operation Hometown, announced that he was calling a meeting of his committee chairmen in the near future to discuss the steps to be taken in trying to influence medical legislation.

The speaker of the evening, Bernard Kliman, M.D., who is an associate in medicine at Harvard Medical School and a director of the Steroid Research Laboratory of the Endocrine Unit at the Massachusetts General Hospital, was introduced by Dr. Denison. Dr. Kliman spoke on the investigative procedures in pituitary and adrenal disorders. He used kodachrome photographs to illustrate the various changes seen in pituitary and adrenal disorders and gave a clear-cut discussion of the tests now currently used to investigate these disorders.

EARLE M. DAVIS, M.D.
Secretary

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held on January 19, 1965 at The Ledges in Wiscasset, Maine.

The Nominating Committee presented a slate of officers for the year 1965 which was unanimously approved.

President, Carl R. Griffin, Jr., M.D., Boothbay Harbor
Vice-President, Alan W. Zeller, M.D., Damariscotta
Secretary-Treasurer, George W. Bostwick, M.D., Newcastle
Delegates to the Maine Medical Association House of Delegates: Drs. John F. Andrews, Boothbay Harbor and Ralph C. Powell. Damariscotta. Alternates: Drs. Charles E. Burden and Paul A. Fichtner, both of Bath

Edward K. Morse, M.D. of Rockland reported on the recent Council meeting of the Maine Medical Association. The Council feels that although Medicare provisions will undoubtedly be passed by Congress, the Maine Medical Association must go on record pointing out to Congress and to the people of the State of Maine the deficiencies in the proposed bill. The problems of blood banking operations in the state and nation were outlined.

Francis A. Winchenbach, M.D. spoke on possible forms which Medicare will take and how they will relate to voluntary health agencies and medical cost prepayment plans.

Thomas A. Martin, M.D., President of the Maine Medical Association, spoke on the current legislative program.

Carl A. Brinkman, M.D. of Portland, the guest speaker, was introduced by Edward L. Kinder, Jr., M.D. Dr. Brinkman spoke on brain scanning with illustrated slides.

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on February 16, 1965. The meeting was called to order by the President, Carl R. Griffin, Jr., M.D.

Ferris S. Ray, M.D. of Portland, guest speaker of the evening, was introduced by Dr. Griffin. Dr. Ray discussed the surgery of vein grafting, with pictorial presentation of saphenous-vein arterial grafting in the leg.

GEORGE W. BOSTWICK, M.D.
Secretary

YORK

Twenty-two members and guests were present at the annual meeting of the York County Medical Society which was held on January 13, 1965 at the Goodall Hospital in Sanford, Maine. Following a social hour and dinner, the meeting was called to order by the Vice-President, Alvin A. Hoffman, M.D. in the absence of the President, Roger J. P. Robert, M.D.

The Nominating Committee presented the following slate of officers for the year 1965 which was approved:

President, Alvin A. Hoffman, M.D., York
Vice-President, Walter R. Peterlein, Jr., M.D., Springvale
Secretary-Treasurer, Charles W. Kinghorn, M.D., Kittery
Delegates to the Maine Medical Association House of Delegates: Drs. Carl E. Richards, Sanford, Marcel P. Houle, Biddeford and Robert F. Ficker, Kennebunkport. Alternates: Drs. G. Patrick Shaw, Biddeford, Stephen A. Cobb, Sanford and Kenneth E. Leigh, York
Executive Committee, Drs. Alvin A. Hoffman, Walter R. Peterlein, Jr., Charles W. Kinghorn, Carl E. Richards and Kenneth E. Leigh
Censor Committee, Drs. Stephen A. Cobb, Melvin Bacon, Sanford and Willard H. Bunker, York Harbor

Paul S. Hill, Jr., M.D., Councilor for the First District, presented a brief report relative to Association matters.

Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association, gave a very fine talk on state and national matters.

Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, spoke relative to Blue Cross-Blue Shield.

CHARLES W. KINGHORN, M.D.
Secretary

Letter to the Editor

Daniel F. Hanley, M.D., Editor
The Journal of the Maine Medical Association

Dear Doctor Hanley:

Our article, "Folic Acid Deficiency in Pregnancy: A Sorely Neglected Clinical Entity" appeared in the December, 1964 issue of your journal.

We admitted rushing into print hastily because of the gravity of the problem of this fulminating anemia of pregnancy. We have had occasion to regret only one statement. Since most readily available prenatal capsules contain 1 mg. folic acid, these were recommended. The hazard of masking the rare case of true pernicious anemia occurring in pregnancy seemed less than that of pregnancy without folic acid prophylaxis. An effective but not dangerous daily dose of folic would be 400 mcg.

Prominent hematologists are applying leverage to manufacturers to furnish prenatal capsules with this safe but effective dose of folic acid. Better labelling is to be hoped for. One manufacturer has a prenatal capsule — (without folic acid), — F (folic acid), — F1 (Fluoride), and another — OT with a bowel softener to compound the confusion.

Yours sincerely,
MASON TROWBRIDGE, JR., M.D.
GEORGE O. CHASE, M.D.
ANDERS T. NETLAND, M.D.

News, Notes and Announcements

Postgraduate Course in Clinical Cardiology
Central Maine General Hospital
Lewiston, Maine
Co-sponsored by the
Central Maine General Medical Staff and
The Maine Heart Association, Inc.

Hiebert Hall 3:00-5:30 P.M.

March 24, 1965 "Diagnosis and Treatment of Cardiac Arrhythmias"
Lawrence Ellis, M.D.

March 31, 1965 "Recognition and Management of Coronary Heart Disease"
David Littmann, M.D.

Fee: $25.00.
No fee for interns or residents.

The Children's Hospital of Philadelphia
and
The Department of Pediatrics, School of Medicine, University of Pennsylvania
ANNOUNCE
A Five-Day Refresher Course in Pediatrics
For Pediatricians and General Practitioners
MAY 24 — MAY 28, 1965

The Curriculum will consist of Lectures and Clinics, given by members of the Faculty Staff, in selected aspects of Contemporary Pediatrics in which important advances are being made
TUITION IS $150.00

Registration and Inquiries should be sent to:
Children's Hospital of Philadelphia
Postgraduate Education Committee
1740 Bainbridge Street, Philadelphia, Pa. 19146

Make Checks Payable to:
The Children's Hospital of Philadelphia

Department of Health and Welfare
Division of Maternal and Child Health
Including Services for Crippled Children
(By Appointment Only)

Orthopedic Clinics

Bangor — Eastern Maine General Hospital
9:00 a.m. and 1:00 p.m.: Jan. 21, Mar. 25

Cardiac Clinics

Bangor — Eastern Maine General Hospital
9:00 a.m.: Jan. 22, Feb. 12, 26, Mar. 12, 26
Portland — Maine Medical Center
9:00 a.m.: Every Friday (Holidays excepted)

Clinics for Mentally Retarded Preschool Children

Waterville — Thayer Hospital
9:00 a.m.: Jan. 20, Feb. 3, 17, Mar. 3, 17, 31

Adolescent Clinics

Portland — Maine Medical Center
1:00 p.m.: Jan. 27, Feb. 24, Mar. 24

Pediatric Clinics

Bangor — Eastern Maine General Hospital
1:30 p.m.: Jan. 22, Feb. 26, Mar. 26
Fort Kent — Peoples Benevolent Hospital
10:00 a.m.: Mar. 24

Boston Medical Reports

Tues., Mar. 23 — 12:00 Noon
(Repeated Thurs., Mar. 25 — 10:05 P.M.)
Problems in the Management of Graves' Disease
Sidney H. Ingbar, M.D.
Harvard Medical School
Lewis E. Braverman, M.D.
Tufts University School of Medicine

Tues., Mar. 30 — 12:00 Noon
(Repeated Thurs., Apr. 1 — 10:05 P.M.)
Modern Immunization Procedures in Children and Adults
Sydney S. Gellis, M.D.
Boston University School of Medicine
Geoffrey Edsall, M.D.
Harvard School of Public Health
Samuel Katz, M.D.
Harvard Medical School

Tues., Apr. 6 — 12:00 Noon
(Repeated Thurs., Apr. 8 — 10:05 P.M.)
Peritoneal Aspiration as an Aid in Diagnosis of Abdominal Disease
Ralph C. Richards, M.D.
University of Utah College of Medicine

Tues., Apr. 13 — 12:00 Noon
(Repeated Thurs., Apr. 15 — 10:05 P.M.)
Cancer Chemotherapy — Practical Considerations
Sidney Farber, M.D.
Harvard Medical School

Albany Two-Way Radio-TV Conferences

Wed., Mar. 24 — 12:00 Noon
Surgery of the Small Intestine
John A. Balint, M.B., Charles Eckert, M.D.
Albany Medical College

Unanswered

From Albany Two-Way Radio-TV Conference, Wed., Feb. 17, 1965.

Question: Role of Mg. in Muscle Spasm?

Answer: Disorders of the Motor Unit Which May Produce Transient Paralysis.

David Grob, M.D. and Richard J. Johns, M.D. Proc. of A.R.N.M.D. *38* pp. 601-621, 1958.

"Reduction in the plasma concentration of magnesium is reported to cause muscle spasm and convulsions, resembling tetany, probably due to increased excitability of nerve and muscle.

"Increase in plasma concentration following administration of large doses of magnesium salts produces muscular weakness, as well as depression of the central nervous system and vasodilatation, with reduction in blood pressure."

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine
November 10-12, 1964

THROUGH EXAMINATION

Iraj Assefi, M.D., Benedictine Hosp., Kingston, N. Y.

Umit C. Ergin, M.D., Gowanda State Hosp., Helmuth, N. Y.

Kazem Fani, M.D., 351 Myrtle Ave., Albany, N. Y.

Joseph P. Fanning, M.D., 28 Dietz Rd., Hyde Park, Mass.

Julia M. Fusco, M.D., 105 West 13th St., New York, N. Y.

Necdet Gureli, M.D., 19 Lyndon St., Caribou, Me.

Vassilios Handanos, M.D., Pineland Hosp. & Training Ctr., Pownal, Me.

Santiago Leyte-Vidal, M.D., Pineland Hosp. & Training Ctr., Pownal, Me.

Javad Mashkuri, M.D., 11 Peterson Ave., Caribou, Me.

Henry J. Messawer, M.D., 201 Saint Paul St., Brookline, Mass.

Ioan Metianu, M.D., 424 East 77th St., New York, N. Y.

Livio H. Rev-Kury, M.D., 165 Longwood Ave., Brookline, Mass.

Reginald D. Smith, M.D., 5 Suburban Rd., Worcester, Mass.

Waclaw R. Wolodzko, M.D., Pilgrim State Hosp., West Brentwood, N. Y.

Michael P. Yazitzis, M.D., Good Samaritan Hosp., Cincinnati, Ohio

THROUGH RECIPROCITY

Horace R. Allison, Jr., M.D., Presque Isle, Me.

Irvin Dorfman, M.D., 176 Colonial Pkwy, Yonkers, N. Y.

Prescott Jordan, Jr., M.D., 772 Fisher Bldg., Detroit, Mich.

John R. Kane, M.D., 70 Arbor St., Wenham, Mass.

Virginia A. Long, M.D., 12 Fleet St., Portsmouth, N. H.

Cecil J. Metcalf, M.D., Bangor, Me.

John R. Stuart, M.D., 97 Seavey St., Westbrook, Me.

Orvar Swenson, M.D., 707 W. Fullerton Ave., Chicago, Ill.

Foster L. Vibber, M.D., 278 Highland St., Worcester, Mass.

Philip J. Villandry, M.D., 393 Spring St., Portland, Me.

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Wegener's Granulomatosis
Diagnostic Difficulties—Case Report

L. Fishman, M.D. and M. A. Chapin, Ph.D., M.D.*

Wegener's Granulomatosis is an uncommon, but not unusually rare condition characterized by extensive granulomatous infiltration of the respiratory tract and widespread systemic involvement. The disease-complex was first reported by Klinger in 1931,[1] but formulated by Wegener in 1936.[2] Clinically it is regarded as a triad of sinus disease, pulmonary infiltrations and nephritis, however, it can be more properly considered a disseminated process in which profound constitutional symptoms of arthritis, skin lesions, peripheral neuropathy, ocular manifestations and otologic signs are also clinical findings. The symptoms include those of fever, chills, sweating, a persistent sinusitis or rhinitis, cough, dyspnea, hemoptysis, anorexia, nausea, vomiting, abdominal pain, weakness, weight loss, arthritic pains and ear ache. Physical findings depend on the systems involved. The sinusitis may be severe enough to cause ulcerations of the bony or cartilaginous structures; the lower respiratory tract may show wheezing, rhonchi, musical rales, diminished tactile and percussion fremitus; the eyes may show an episcleritis. Many cases show clinical and laboratory evidence of renal involvement such as edema, red blood cells and white blood cells in the urine, casts, albumin, azotemia and uremic symptoms and signs especially late in the disease.

Radiographically, the organs of the respiratory tract are the only ones showing lesions. One or both lungs may be involved. The lesions are usually well-defined rounded opacities ranging in size from a few to a number of centimeters in diameter. Cavitation due to central necrosis is common. The findings may suggest neoplasm or tuberculosis. The paranasal sinuses, the nasal mucosa and nasal cartilages are always involved to some extent. This includes mucosal thickening in the antra. Destruction of bone and nasal cartilages may occur.

Pathologically, the lesions consist of a necrotizing granulomatosis characterized by aseptic granulomas and striking vascular lesions. In the lungs, the circumscribed nodules may vary up to 5 centimeters or more in diameter, often with soft friable necrotic centers. These nodules are often situated adjacent to ulcerating lesions of the smaller bronchi and bronchioles. Histologically, the granulomas reveal a central coagulation necrosis surrounded by a zone of inflammatory cells containing polymorphonuclear leukocytes, lymphocytes, plasma cells, histiocytes, fibroblasts and multinuclear giant cells. The vascular alterations consist of a localized necrotizing inflammation of both arteries and veins with fibrinoid changes. The vascular lesions may involve other structures especially the kidneys and spleen. The kidneys may show a focal necrotizing thrombotic glomerulitis, which is more or less widespread. In the subacute phase these lesions show a tendency to heal by capsular epithelial proliferation and granuloma formation. In addition, focal granulomas may be encountered in the renal interstitium. The lesions may range from small foci of necrosis to total infarction.

The etiology of the disease is unknown, treatment only palliative at best and the prognosis poor, eventually terminating in death from respiratory failure and uremia. Several excellent reviews have described it as an entity, the most recent being that of Fred, et al.[3] More than 100 case reports, including seven in the November 1964 Index Medicus, have appeared in the medical literature, attesting to the fact that it is not as rare as formerly believed.

The purpose of this paper is to report another instance of this disease and to emphasize the difficulties encountered in its diagnosis and treatment.

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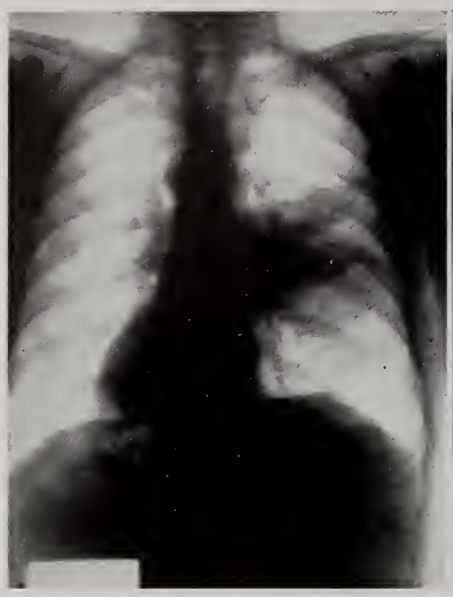


Fig. 1. Initial postero-anterior roentgenogram of chest showing infiltration involving upper portion of lower lobe and lower portion of upper lobe.



Fig. 2. X-ray film one week later showing progression of infiltration in spite of antibiotic therapy.



Fig. 3. X-ray film two weeks after intensive antibiotic therapy, showing progressive infiltration.

Case Report

A 55-year-old chicken farmer was first admitted to the Central Maine General Hospital 9-1-63 complaining chiefly of upper abdominal pains which he had had for a week associated with nausea, vomiting, fever, chills, and cough. In August, about one month previously, he had begun to feel tired but had continued working. Then, 10 days prior to admission, he began to have nausea with occasional emesis of ingested foods. He also started to have a cough with daily fever and chilliness. He was given penicillin injections and then Declomycin® orally, but these had no effect and he continued having a moderately productive cough, raising mucoid to greenish sputum, with continued fever and chills. There was some soreness in the chest on breathing and on coughing, but no definite pleuritic pain. Slight hemoptysis had occurred on one or two occasions. He had lost some 10 lbs. in weight.

In his past history, he had had bronchial asthma since the age of six months though this had been much less marked since the age of 10 years. He was born and raised on a farm where both general farming and orcharding were emphasized. He started in the chicken business upon graduating from high school at the age of 16 and worked with his father on all other farm occupations including the spraying of orchards, dairying, and care of farm animals. He began smoking at the age of 22. For the past 15 years, he had been raising broilers and at the time of this illness was keeping some 26,000 on hand. He had tonsillitis at the age of 35 and again at the age of 44 at which time his bronchial asthma again began to become bothersome. He stopped smoking then and had never resumed this. For many years, he had occasionally had sick headaches lasting several hours to a day and associated with nausea and vomiting. There was a family history of bronchial asthma as well as other allergies.

His temperature was 101.4° by mouth, pulse 112, respirations 24, blood pressure 120/80. He appeared acutely ill, was coughing frequently, raising but little, and was weak and somewhat listless. The skin was quite warm and dry. There was moderate malar telangiectasia. The eyes, ears, and nose were not remarkable. There was slight pharyngeal redness. The neck was supple. There was no adenopathy. The heart was of normal size, position, and rhythm and there were no murmurs. The chest was emphysematous with increased AP diameter. Throughout both lung fields expiratory wheezes and musical sounds could be heard. There was slight dullness at the right midlung field posteriorly and laterally and breath sounds seemed slightly diminished in these areas. The liver edge was palpable just below the right costal margin. There was suggestive epigastric tenderness to pressure. No masses, spasm, or enlargement of viscera were noted. The extremities were normal as were rectal and neurological examinations.



Fig. 4. Right pneumonectomy -9/8/63. Posterior surface of lung. Irregularly lobulated and mottled patches of emphysema, pneumonitis and atelectasis. No thrombi.

The hemoglobin was 14.1 gms.% with an hematocrit of 45%. The white cell count was 7800 with 79 segmented neutrophiles, 10 band forms, 6 lymphocytes, and 5 monocytes. The urine was yellow, hazy, and acid; the specific gravity was 1.021; a slight trace of albumin was present, but no sugar or acetone and the sediment was negative. The BUN was 34.4



Fig. 5. Cut surface. Large patches of dense, yellow, healed pneumonitis, interspersed with bright crimson lobules of active pneumonitis. Emphysema and atelectasis. Anthracosis minimal. No major thrombosis.

mgs.%. The FBS was 115 mgs.%. The serology was negative. The serum total bilirubin was 1.3 mgs.%. Two sputum cultures showed a few colonies each of alpha-hemolytic Streptococci, Neisseria, and B. subtilus. A stool culture grew E. coli and Proteus. The cold agglutinins were negative in all dilutions. No acid-fast bacilli were found in several sputa. The alkaline phosphatase was 13.2 Bodansky units. After adequate hydration, the BUN was 18.2 mgs.%. An ECG showed sinus tachycardia, but was otherwise within normal limits. X-ray films of the chest showed increased densities in the right lung posteriorly involving the apical portions of the right lower lobe and the base of the right upper lobe, consistent in appearance with an acute pneumonic process. (Fig. 1) Reexaminations of the chest 9-10-63 and 9-16-63 showed further progressive involvement of the consolidations previously noted. (Fig. 2, 3) The left lung was clear. No evidence of cavitation in the area of involvement could be seen. Bronchoscopy, accomplished 9-11-63, showed the mucosa of the right main-stem bronchus to be hyperemic and bleed easily; otherwise, nothing abnormal was seen in any area. A biopsy of the right main-stem bronchus was taken and secretions removed for culture and Pappanicolau smear. The latter test was negative. The biopsy tissue showed nests of small hyperchromatic epithelial cells in broad irregular medullary sheets showing well-defined anaplasia and occasional mitotic figures, and an initial impression was that this represented a highly undifferentiated epidermoid carcinoma.

On 9-18-63, a right pneumonectomy was accomplished. This was complicated by excessive bleeding from the right pulmonary artery and 11 units of blood were used in replacement therapy during this surgery. He recovered slowly from this and it was obvious that his previous toxic course with chills and fever had been relieved.

The lung which was removed showed two large masses, one in the upper portion of the lower lobe, the other in the lower portion of the upper lobe. Both were quite firm and somewhat irregular in outline. (Fig. 4, 5) Histologically, in multiple sections, this showed a necrotizing granulomatous process in the circumscribed inflammatory masses which, grossly, presented firm, grey surrounding zones and soft red centers. Numerous polymorphonuclear leukocytes, lymphocytes, plasma cells

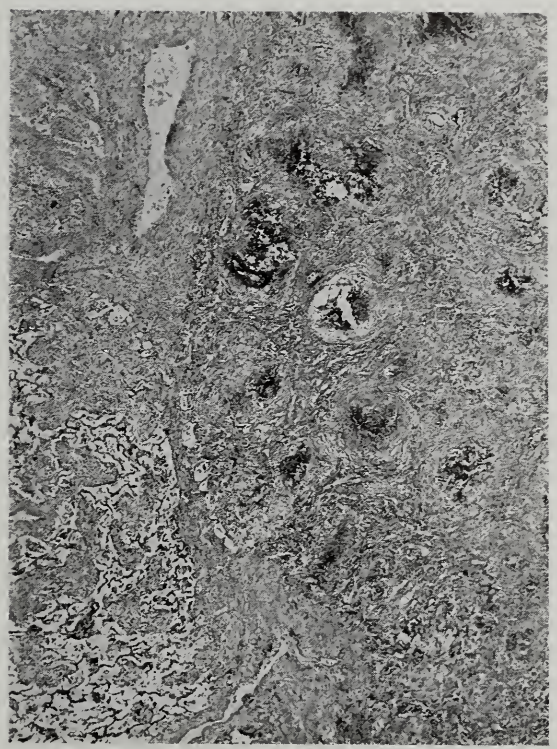


Fig. 6. Interlobular septum; one side nonactive healed pneumonitis; other side very active granulomatous pneumonitis. Each circumscribed dark area is a focus of granulomatous pneumonitis about a vessel in a state of acute vasculitis as in Figure 7. X-40 H&E.

and occasional eosinophils, as well as multinucleated giant cells of foreign body and Langhan's type were present. Hemorrhagic infarcts with arteries occluded with thrombi were noted. (Fig. 6, 7, 8) Since psittacosis was suspected clinically, these findings were noted to be similar, and this was initially the impression. His subsequent hospital course was one of steady slow improvement and he was finally discharged 10-24-63.

Second Admission 5-25-64, for extraction of carious and infected teeth. From the time of his last discharge, he had been rather constantly bothered with nausea and occasional vomiting. Thorazine® relieved the nausea. He had also had constant trouble with his nose in that, intermittently, large crusts would form and, after several days, there would be severe nasal bleeding. This had gradually become increasingly marked and he was seen by otolaryngologists for purposes of fulguration. He was told that there were ulcers present in the mucous membranes of his nose causing the crusting and bleeding. In addition, one month prior to this admission, his right eye had become red and painful. This was diagnosed as an episcleritis by his opthalmologist and was treated with eye drops containing a cortisone derivative, which served to clear up the eye symptoms. Because of all these continued symptoms, his family physician had thought that it might be his infected teeth which were at fault and he had been advised to have them extracted. He had had no chills or fever, but had continued to lose some weight. On physical examination, the vital signs were normal and the blood pressure was 110/70. He was somewhat thin and preferred to wear dark glasses because of the light hurting his eyes. There was moderate scleral injection of both eyes more marked on the right. The fundi were normal. There was no adenopathy. The left lung was clear and resonant. The heart, abdomen, and remainder of the examination was within normal limits. The hemoglobin was 12.3 gms. and hematocrit 35%. The white cell count was 7700 with 73 segmented neutrophiles, 3 band forms, 12 lymphocytes,



Fig. 7. Septal arteriole in advanced state of occlusive acute vasculitis. X-300 H&E.

7 monocytes, and 5 eosinophiles. An ECG was within normal limits. X-ray films of the chest showed moderate emphysema of the left lung. The BUN was 30 mgs.% and urinalysis showed specific gravity of 1.014, with 30 mgs.% albumin and 10-12 WBC in the sediment.

The next day, sixteen remaining teeth were extracted. These were noted to be carious and slightly infected at the roots. His subsequent course was not remarkable and he was discharged two days later.

Third Admission 7-2-64. Following his return home after extraction of teeth, he had continued to have progressive listlessness, anorexia, and frequent nausea. The latter was somewhat controlled with Thorazine® administration. However, the anorexia became so marked that he was eating but little and had lost 18 pounds during this period of time. He had continued to have intermittently the formation of crusts in his nose with subsequent nasal bleeding and had required occasional fulguration of the nasal mucous membranes because of this. Also, two weeks before this admission, his left ear had rather abruptly become swollen, red, very tender and painful. This was thought to be due to an infection and he had been given Declomycin.

His temperature was 98.6° orally, pulse 106, respirations 24, blood pressure 90/62. His weight was 127 pounds. He was noted to be thin and cachectic and appearing chronically ill and moderately pale. The right ear lobe was moderately thickened, erythematous, and somewhat tender. The sclerae were now nearly clear. There was no adenopathy of note. The chest was emphysematous. The left lung field was clear and resonant. The heart was again normal with a regular rhythm and no murmurs. The liver edge descended 2 cm. below the right costal margin on inspiration. The spleen was not palpable. The remainder of the examination was within normal limits.

The hemoglobin was 9.6 gms.% with hematocrit 31.5%. The white cell count was 7100 with 72 segmented neutrophiles, 4 band cells, 16 lymphocytes, 5 monocytes, and 3 eosinophiles. Several urinalyses showed relatively fixed specific gravity of 1.012 and albumin 100 to 300 mgs.% present.

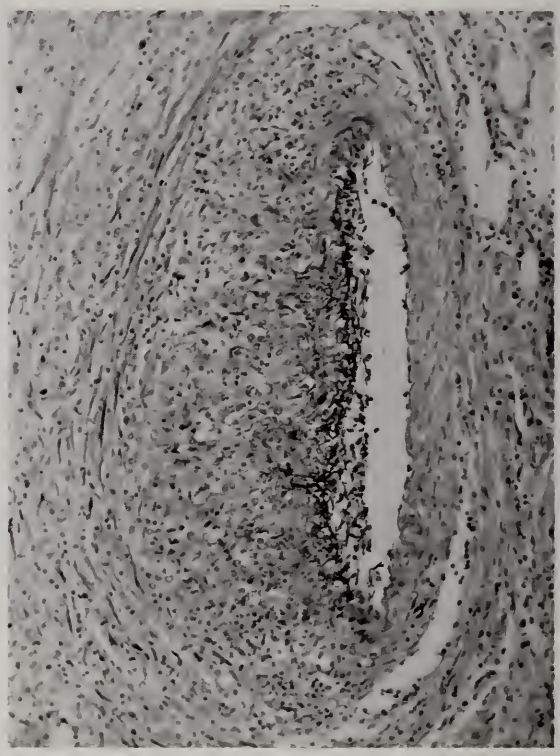


Fig. 8. Septal arteriole: one half reasonably uninvolved: opposite side in advanced state of organizing vasculitis. Note the acute inflammatory infiltration at either end of the mural thrombus, suggesting that the process may be spreading circumferentially about the vessel. X-300. H&E.

The sediment showed 15-20 WBC, over 100 RBC, and rare coarsely granular casts. The BUN was 70.5 mgs.%. The serology was negative. The sedimentation rate was 30 mm./hr. A urine culture grew out E. coli in significant numbers. The serum proteins were 5.4 gms.% with albumin 1.8 and globulins 3.6 gms.%. The stools were guaiac negative. X-ray films of the chest showed no change. X-ray films of the sinuses showed no evidence of infection. A retrograde pyelogram showed normal calyxes, pelves, and ureters on both sides. A routine serum paper electrophoresis showed a gammaglobulin of 0.54 and albumin of 1.68 gms.%. There were moderate increases in both the beta and alpha-2 globulins.

Since it was now apparent that he had rather severe chronic granulomatous disease with severe renal impairment appearing after a fluctuant course of involvement of the lung, eyes, ears, sinuses, and nose, treatment was started with Medrol® 8 mgs. four times a day on alternate days, in addition to multiple vitamins, Thorazine for the nausea, and antibiotics for the urinary tract infection. There was quick response to these measures with improvement in appetite and sense of well being. The right ear cleared completely and there was no further crusting and bleeding from the nose, and, by the time of his discharge 7-24-64, he was eating well and much stronger. At this time, his BUN was 76 mgs.% and urinalysis still showing 100 mgs.% albumin, numerous red and white cells, and casts in the sediment.

In the interim, since this last discharge, he has continued to take all medications and had gained some 20 pounds in weight. He is active and working as an insurance agent. The last BUN in January 1964 was 32 mgs.% and urinalysis continued to show presence of protein, cells, and casts.

Discussion

Early in the course of this case, the diagnosis was

very illusive. Had one thought of the possibility of Wegener's Granulomatosis, perhaps it would have saved a great deal of concern and anxiety although it would not have solved our immediate problem. The patient's symptoms were fulminating and his life was considered to be in jeopardy. The only tangible findings at the time were the infiltrations in the right lung. The etiology of this infiltration could not be determined from the x-ray. The increased densities in the apex of the lower lobe and the base of the upper lobe of the right lung were felt to be consistent with an acute pneumonic process. This process, however, did not respond to antibiotic therapy and, in fact, showed a progressive involvement of the lungs. The etiology was even more confused by a bronchial biopsy report suggesting a highly undifferentiated epidermoid carcinoma. On the basis of this report and the severity of the patient's symptoms which were unresponsive to medical therapy, a pneumonectomy was done. Following this the toxic symptoms disappeared, but the patient's convalescence was prolonged. The histology of the resected specimen was reported as that of a necrotizing granulomatous process of an unknown etiology. A review of the patient's history and the pathology found suggested to us the possibility of psittacosis. The compliment fixation test did not confirm this suspicion; consultative surveys of the histologic sections by several pathologists were not in agreement and the usual case of psittacosis would have showed an alveolar and interstitial type of pneumonitis rather than the granulomas. The bloody crusting in the patient's nose was the next thing that showed up followed by episcleritis, inflammation of the ear cartilage and finally renal involvement. It was only at this stage of the disease that a diagnosis of Wegener's Granulomatosis was entertained.

A variety of other conditions which present necrotizing and granulomatous processes could be considered in a differential diagnosis. Specific infectious granulomatous diseases such as tuberculosis, lues, tularemia, blastomycosis or histoplasmosis have etiologic organisms which can be isolated in the acute stage. Their histological appearances are also differentiating factors. Conditions of vascular allergy such as Loeffler's pulmonary infiltrations are not associated with the typical signs and symptoms of Wegener's syndrome. Boeck's sarcoid which can and does involve many of the organs attacked by Wegener's Granulomatous disease is non-necrotizing in character. Idiopathic periarteritis nodosa differs from Wegener's syndrome in that it is usually seen in older people; there is no antecedent necrotizing sinusitis or pulmonary granulomatosis; the lungs in this condition show changes only in the arteries with nodular aneurysms visible along the course of the medium sized arteries, and this is not the case in Wegener's disease.

Allergic periarteritis nodosa offers a real challenge as a differential diagnosis. However, in this condition there is a family or personal history of allergy which is absent in Wegener's disease. Sinus disease frequently present in allergic periarteritis nodosa is never as severe as in Wegener's disease. The invasiveness and frequency of necrotizing granulomatous lesions in the respiratory tract are never as marked in allergic periarteritis nodosa. Renal lesions and the severity of such is greater in Wegener's disease. Eosinophilia, so prominent in allergic periarteritis nodosa, is only occasionally seen in Wegener's disease. In spite, of these differential points, some writers consider Wegener's Granulomatosis as a special form of periarteritis nodosa while others have looked upon it as an individual disease entity.

Summary

1. Wegener's Granulomatosis is a disease entity consisting of necrotizing granulomata and angiitis involving the sinuses, lower respiratory tract, kidneys and other organs.

2. A case report of this disease is documented in detail.

3. The sequence of events leading to the diagnosis in the case presented is discussed.

4. Differential points from other granulomatous diseases and arteritis conditions are mentioned.

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Steroid Ulcers

Report of 28 Cases with Review of Literature and Discussion of Pathogenesis

FENNELL P. TURNER, M.D.*

For more than a decade it has been recognized that gastroduodenal hemorrhage and/or perforation occasionally occur as complications of therapy with either pituitary corticotropin or adrenal cortical steroids. The pathogenesis of such "steroid ulceration" remains obscure, however, and some continue to doubt its actual existence. For these reasons it is believed that a review of the literature and a report of additional clinical examples of this entity will be of interest.

CLINICAL MATERIAL

It was found on reviewing our clinical records covering the period from January, 1951, through December, 1963, that 28 patients had developed gastroduodenal ulcerations while undergoing treatment with ACTH or adrenal steroids (Table 1). Of these 28 patients, 20 denied having had significant gastric symptoms prior to their steroid therapy. Seven of the latter group had had negative X-ray findings on "Upper G.I. series" done immediately before or soon after beginning steroid therapy. There were 8 patients with past histories of more or less epigastric distress relieved by food or antacids (Table 2). In 2 of these cases a diagnosis of peptic ulcer had been made in the past, but both were asymptomatic at the time the steroid therapy was instituted. In one case the diagnosis of ulcer had been based upon X-ray studies made 29 years previously; the other patient had had a gastrectomy done for proven duodenal ulcer several years before. The latter had had no further ulcer symptoms until he developed them during the course of steroid therapy, when an active marginal ulcer was demonstrated by X-ray.

In addition to these 28 patients in whom gastroduodenal ulceration was proven, there were 12 others who developed dyspeptic symptoms strongly suggestive of peptic ulcer, one of whom even had melena. In none of this group, however, was ulcer actually demonstrated by either surgery or X-ray.

Of the 28 cases with verified ulcers, 9 were first diagnosed at autopsy, 14 were discovered during X-ray studies, and 5 were confirmed by surgery (Table 3). The 14 nonoperative cases were all treated successfully by medical measures; these consisted of the usual dietary and antacid regimens plus reduction or elimination of the steroid preparations. In 5 cases these agents were discontinued altogether; in 4 cases the dosage was decreased to 50 mg. of cortisone (or its equivalent) daily; in 2 cases the dose was reduced to 12.5 to 25 mg. daily; the other 2 cases have been treated intermittently with short periods of 50 to 100 mg. daily and none during the intervals between such courses.

Of the 5 cases in whom diagnosis of ulcer was confirmed at operation, 3 presented massive gastroduodenal hemorrhage as the initial symptom and all required

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TABLE 1

SYMPTOMS IN 28 PATIENTS WITH STEROID ULCERATION

	LOCATION OF ULCER				
	Duodenal	*Gastric*	*Duodenal & Gastric*	*Marginal*	*Esophageal*
Pain-dyspepsia	6	3	1		
Bleeding	3	1	2		
Bleeding & pain	2		1	1	1
Perforation	4				
Perforation & bleeding		1			
None, found at autopsy	1				
None, found on G.I. series		1			
TOTAL	16	6	4	1	1

TABLE 2

POSITIVE PAST HISTORY VERSUS NEGATIVE PAST HISTORY

		Duodenal	*Gastric*	*Duodenal & Gastric*	*Marginal*	*Esophageal*	*Total*
Positive History	X-rays not done or X-rays negative	4	1			1	6
	X-rays positive	1*			1**		2
Negative History		11	5	4			20
		16	6	4	1	1	28

*G.I. series 29 years ago had shown evidence of duodenal ulcer. Patient free of symptoms at start of steroid therapy.
**PGE 4½ years ago for duodenal ulcer with pyloric obstruction. Flare-up of ulcer symptoms with marginal ulcer by X-ray after steroid therapy for 4 months.

TABLE 3

MANAGEMENT OF STEROID ULCERATION

		Duodenal	*Gastric*	*Duodenal & Gastric*	*Marginal*	*Esophageal*	*Total*
Negative Past History	Medical Rx	8	3	1	0	0	12
	Surgical Rx	0	1	1	0	0	2
	Found at autopsy	3	1	2	0	0	6
	Total	11	5	4	0	0	20
Positive Past History	Medical Rx	1	0	0	1		2
	Surgical Rx	1	1	0		1	3
	Found at autopsy	3	0	0			3
	Total	5	1	0	1	1	8
	Grand Total	16	6	4	1	1	28

emergency surgery. One of these patients died. Another was found to have, in addition to massive bleeding, a walled-off perforation which had created an abscess containing several hundred cc. of pus within the lesser peritoneal sac. The third patient with bleeding was found to have peptic esophagitis associated with a hiatus hernia.

Of these 28 patients whose ulcers first became manifest during steroid therapy, 14 were receiving long-term treatment for rheumatoid arthritis; 2 were being treated for psoriasis associated with arthritis; one patient was under treatment for each of the following conditions: acute rheumatic fever, asthma, ileocolitis, chronic metatarsal bursitis, serum hepatitis, osteitis pubis, chronic uveitis, acute monocytic leukemia, fibrosarcoma, postpneumonectomy empyema with presumed adrenal failure, Buerger's disease with urticaria caused by penicillin sensitivity and third-degree burns also complicated by urticaria from penicillin therapy.

Seven of the 28 cases had received ACTH only; 16 had received corticosteroids only; 5 had been treated with both ACTH and steroids. In 2 of the cases the steroid therapy was of such brief duration that some doubt was expressed of its being a factor. However, these cases are being included for the sake of completeness. One of these was the patient with Buerger's disease, who had received penicillin for treatment of an infection of an amputation stump and thereafter developed an allergic reaction. For this latter he was given ACTH intravenously for 3 days and then suddenly, with no prodromal symptoms other than a brief episode of nausea and vomiting, perforated an acute duodenal ulcer. The other patient, after receiving a total dosage of 150 mg. of cortisone over a 36-hour period, expired from massive gastroduodenal hemorrhage. Steroid therapy had been given in treatment of urticaria presumably due to penicillin therapy. Autopsy examination of this patient with 40% second and third-degree burns and associated delirium tremens, revealed a large acute duodenal ulcer. There were also 3 small acute gastric ulcers.

With the exception of these last 2 patients, the shortest duration of therapy in the case of corticosteroids was 11 days. During this period this patient received from 300 to 800 mg. of cortisone per day for a total of 7,300 mg. With ACTH the shortest duration of therapy was in a patient who had received 40 to 80 units of ACTH gel daily for 12 days for a total of 760 units. In contrast with these 2 patients the remaining 24 patients had received therapy in varying doses for as long as 10 years in the case of corticosteroids and as long as 4 years in the case of ACTH. The dosage schedule for corticosteroids in the period just prior to onset of ulcer was 100 mg. or more of cortisone per day or its equivalent in 9 patients, 75 to 100 mg. per day in 4 patients and 40 to 100 mg. per day for varying periods of time in 5 patients. One patient had received only 25 mg. per day. This patient was a 73-year-old poorly nourished male who had been taking Medrol, 2 mg. b.i.d., for one month prior to admission because of arthritis of the spine. This patient was the one mentioned previously who had given a history of duodenal ulcer with bleeding and a positive X-ray diagnosis 29 years before. Following this present admission for weakness, anemia, and severe melena of one week's duration, a subtotal gastric resection had been carried out. Three days after operation the patient died in acute renal failure. There had also been cardiac decompensation, and post-mortem studies had revealed evidence of marked myocardial fibrosis. Study of the resected specimen in this patient revealed the presence of 2 acute duodenal ulcers, each of which was associated with minimal fibroblastic reaction.

Discussion

Incidence. Neither the incidence of steroid ulceration nor the incidence of ordinary clinical peptic ulcer is exactly known. From clinical and autopsy studies and also from surveys conducted by insurance companies it is believed that peptic ulceration probably occurs at some time during their lifetime in from 5 to 14% of the total population.[1,2] It is also believed that from one to 3% of all people over the age of 20 probably have peptic ulcer at any one time. Incidence of gastroduodenal ulcer in persons with rheumatoid arthritis is somewhat higher and has been estimated at from 4 to 9%.[3,4,5] On the other hand in those patients with rheumatoid arthritis who have been under therapy with ACTH or the adrenal cortical steroids, a striking incidence of from 11 to 31% has been reported.[6,7,8,9,10] Despite the increased incidence in rheumatoid arthritis, the incidence of peptic ulceration in patients with ulcerative colitis given steroid therapy has been found to be from one to 2%.[11,12] This is about the same as that found in the general population. The reasons for the difference between these 2 diseases is not known although it is believed by some that nutritional factors may play a role.

Characteristics of steroid ulceration. It is generally recognized that the frequency of complications bears a direct relationship to both the size of the dose and the duration of therapy. Steroid ulcers have rarely developed in patients who have received less than 4 to 5 days of treatment even when high doses have been used. It has long been recognized that due to its catabolic action steroid therapy is more likely to be injurious when given to malnourished or debilitated patients than it is when given to patients in good health. Edema, osteoporosis, spontaneous fractures, negative nitrogen balance, loss of weight, and hypertension, as well as gastroduodenal ulceration, are frequently found where the dosage has been high or where the therapy has been prolonged. There now seems to be fairly general agreement that steroid therapy of more than 50 mg. of cortisone per day or its equivalent carried out for more than 3 months is apt to cause symptoms of some kind. There are excep-

tions, of course, and some robust patients can tolerate larger doses. Smaller doses than this are relatively safe, but it appears that most patients are unable to tolerate a full therapeutic dose of 75 to 100 mg. of cortisone or its equivalent for any appreciable length of time without developing one or more of the complications of this therapy, among which gastroduodenal ulceration is an occasional, but dramatic example. Following the discontinuance of steroid therapy, ulcer symptoms as a rule rapidly subside, and the incidence of recurrent ulcer disease in the absence of continued therapy is low. On the other hand if therapy in high dosage is once again resumed, the recurrence rate is high. Patients with a past history of peptic ulcer are prone to reactivation of their ulcers after institution of steroid therapy; consequently, if such therapy is indicated rigid precautions should be undertaken.

From both a gross and a microscopic standpoint steroid ulcers are usually acute and are characterized by a paucity of fibrosis and fibroblastic proliferation. They may vary widely in size and in depth of penetration. Multiple ulcers are commonly found, 6 out of 28 cases in this present series. Whereas many steroid ulcers are characteristically asymptomatic, when symptoms do occur, they are frequently dramatic. Acute free perforation and massive hemorrhage are commonly observed, and the mortality is high. Another characteristic is the frequency of involvement of the stomach. Among the 28 patients in this series there were 6 with gastric ulcer and 4 with both gastric and duodenal ulcer. The greater curvature of the stomach was involved in 4; the posterior wall was involved in 2; multiple ulcerations in the antrum were found in one; and in 3 patients the ulcers were found on the lesser curvature. In 2 of these 10 patients there was associated active duodenal ulceration, and in 2 patients there was evidence of old duodenal-ulcer disease, now healed. In years past it had been said that benign ulcers of the greater curvature were unusual and that when such an ulcer was found on X-ray examination malignant disease should be ruled out. However, in 1963 Palmer[13] found greater-curvature ulcers in 7.1% of 311 patients with benign gastric ulcer. And in a large autopsy series the greater curvature was found involved in 14%.[14] Now, it has been found that the greater curvature is commonly involved in patients who have had steroid therapy[5,1516,17] and this is borne out by the appearance of greater-curvature gastric ulcers in 4 of our 10 patients with gastric ulcer.

Miscellaneous historical data. Although pituitary corticotropin was isolated in 1963[18,19] and cortisone was synthesized in 1946[20] it was not until the discovery in 1949[21] that these substances had dramatic effect in the treatment of rheumatoid arthritis and rheumatic fever that the supply of these pharmacologically potent agents was sufficiently plentiful so that they could be given widespread clinical trial. Symptoms of hyperadrenalism were expected in those patients who received large doses of these potent endocrine products. Sprague had said in 1950,[22] "In most of the clinical conditions which have been favorably influenced by administration of adrenal corticotropic hormones ACTH or cortisone, there is no clear evidence of hormonal deficiency; consequently, it appears that in many of these conditions a state of hormonal excess must be created in order to achieve therapeutic benefits." The development of new peptic ulcers or the reactivation of old ulcer disease, however, was not predicted.

The occasional occurrence of gastroduodenal erosions and acute ulcerations following severe injury, burns, illnesses, operations, and CNS disease had been recognized for more than 100 years, and many theories as to their pathogenesis has been presented. Most recently these lesions had been thought to be related in some fashion to acute adrenal insufficiency. Local areas of hemorrhage and acute ulcerations in the stomachs of adrenalectomized rabbits had been observed by Cioffi in 1905, and similar observations had been made by Mann in 1916 as well as by many others.[1] Demonstration through repeated experiments that acute gastrointestinal-tract erosions will develop after many kinds of physical stress,[23] and Selye's concept of the adaptation syndrome[24] did much to arouse interest in this problem. In 1949 it occurred to the author that presumed adrenal failure and the "alarm reaction" as described by Selye were not, however, sufficient to fully explain the extensive areas of stress ulceration as were often observed on post-mortem examination, nor did they seem to explain in any way chronic peptic ulcer of the stomach or duodenum of the typical clinical varieties. Instead, it seemed likely that other factors such as malnutrition and derangement of certain metabolic processes might be of etiological significance in these larger and more severe lesions. Accordingly, it was predicted at this time that corticotropin or corticosteroids might aggravate peptic ulcer and that their unrestricted use in patients with chronic peptic ulcer would be hazardous. A survey of 29 consecutive cases of Addison's disease which had come to autopsy between the years 1930 and 1946 had been carried out;[25] and it was observed that although erosions and small superficial ulcerations had been noted in 4 patients, in only one patient had a typical chronic ulcer been found. It was thought possible that this ulcer had been present prior to the terminal episode as acute adrenal failure in this particular patient was believed secondary to massive invasion of both adrenal glands by metastatic epidermoid bronchogenic carcinoma. These findings in a negative sense served to confirm the theory. When, at this same time, Ragan and co-workers[26] observed delayed wound healing in patients who were receiving ACTH and when early investigative studies also demonstrated a delay in fibroblastic proliferation and in wound repair in experimental animals,[27] it seemed that this was one mechanism by which adrenal overactivity would exert a harmful effect on the natural course of

peptic ulcer. An experimental study was then undertaken in the Spring of 1950 to determine whether patients with peptic ulcer showed any abnormality in adrenal function as determined by the eosinophile response to the stress of operative surgery and to the subcutaneous injection of either adrenalin or ACTH.[28] The results of these studies were equivocal. Whereas a subnormal response to either epinephrine or ACTH was found in some patients with peptic ulcer, it was noted that the stress of gastric surgery was followed by a satisfactory eosinopenia. This tended to confirm the clinical impression that patients with peptic ulcer disease as a general rule had sufficient adrenal reserve to withstand severe stress.

Several months after the initiation of this study the first report of a perforation of ulcer in a patient receiving ACTH was reported by Beck and co-workers.[29] This was soon followed by a second report of a perforation by Habif, et al.[30] Massive bleeding in a patient on ACTH was reported by Smith.[31] Elsewhere cases had been observed where ulcer symptoms apparently had become aggravated by the administration of ACTH,[32] and it was thought by Spiro and co-workers[33] that this was possibly due to an increased secretion of gastric pepsin. In the months that followed more cases were to be reported including complications of peptic ulcer disease in patients receiving adrenal cortical steroids.

My thoughts as to the double role played by the adrenal cortex in the pathogenesis of gastroduodenal ulcer were published in June, 1951.[25] In this hypothesis it was suggested that, whereas the adrenal steroids as homeostatic agents might be effective in preventing certain superficial lesions often seen as the result of the alarm reaction, the larger and deeper ulcers seen in patients subjected to severe stresses and debilitating diseases, and chronic peptic ulcer itself were related to a breaking down of mucosal resistance and an indolence in healing secondary to adrenal cortical hyperactivity. Several months later Gray and his coworkers[34] also suggested that adrenal hyperactivity was a major factor in the development of peptic ulceration. And in a series of reports he presented evidence to show that this was the result of a hormonal stimulation of gastric secretion. On the other hand several observers thought that ACTH and cortisone would be of value in the treatment of stress ulceration[35,36,37] and would be of no effect, beneficial or otherwise, in idiopathic chronic ulcer disease. Many other observers felt that the occurrence of peptic ulcer complications with steroid therapy was merely coincidental, and this last belief is still widespread. A large number of studies have now been carried out in an attempt to prove or disprove these dissimilar hypotheses, and many experiments have been performed in order to determine the effects of adrenal steroids on gastric physiology. The results of many of these studies have been equivocal.

Adrenal function in peptic ulcer. Although the stress of operative surgery[28] and the intramuscular injection of ACTH gel, or the injection of ACTH intravenously,[38] were found to be followed by a normal eosinopenic response, showing that the ulcer patient had adequate adrenal reserve, the eosinopenic response of patients with peptic ulcer to subcutaneously administered ACTH had been found to be deficient in about 70% of the patients.[28,38] The poor response to subcutaneously injected ACTH had been thought partly due to the extravascular inactivation of the hormone. Plasma hydroxycorticosteroid values have also been determined in patients with peptic ulcer. Whereas Rosenbojim and co-workers[39] have found elevated steroid levels, normal levels have been found by others.[40,41] Determinations of the 24-hour urine excretion of adrenal steroids have yielded similarly conflicting results. A decreased urinary steroid excretion has been found by some,[42,43] and a urinary excretion within normal limits has been found by others.[41,44]

The effects of adrenocortical steroids on gastric secretion. In 1950 Spiro, et al., had observed an elevation in uropepsin excretion in subjects receiving ACTH.[33] This was later followed by observations of the increase in pepsin and free hydrochloric acid.[34,35] In other studies no significant difference was found between the uropepsin values in patients with active duodenal ulcer and in normal subjects.[44] Patients have been described where gastric pepsin has remained unchanged despite an increase in uropepsin.[46] A patient has also been reported who had demonstrated an absence of acid despite an increase in blood pepsin and uropepsin levels.[47] And in one of our patients who had developed a lesser-curvature gastric ulcer in 1953 following ACTH, the fasting gastric analysis showed no free hydrochloric acid. After caffeine the free acid was 8 units and 12 units at the end of one and three-quarters and 2 hours. Hirschowitz[48] found an absence of free hydrochloric acid in 2 patients, and Kammerer[5] noted achlorhydria in 4 patients with steroid ulceration. Although Gray had found the long-term administration of ACTH and steroids to be associated in some subjects with an increase in gastric secretion, short-term administration of these steroids has since been found to have no effect.[49,50]

Results of animal experimentation have been similarly ambiguous. Whereas some studies have been interpreted as showing an increased gastric secretion,[45,48,51,52,53,54] others have found the steroid hormones either to be without any significant pharmacological effect on gastric secretion or have found that they cause an actual reduction in gastric secretion.[55,56,57,58] Much of this and other similarly controversial material, together with some of the foreign-language literature, has been summarized in considerable detail in several critical reviews of the subject such as those by Sandweiss,[59] Kirsner,[60] Spiro and Milles,[61]

and Segal,[62] and it is now generally agreed that although adrenal cortical steroids are not in themselves secretagogues their presence in adequate amounts is necessary for the maintenance of normal gastric function. It has been said in reference to this action, as well as in reference to other bodily activities, that the adrenal steroids exert a permissive[63] or conditioning[64] effect on the body tissues. Gastric function will usually be found depressed in starving and debilitated subjects as will the function of many other tissues including the endocrine glands.[65] Gastric secretion is found depressed in Addison's disease and in the adrenalectomized animal,[66] and chronic peptic ulcer is not found in patients with untreated Addison's disease. In those patients maintained on adrenal steroids gastric secretion has, however, been found to return to normal, and steroid ulceration has been described in a number of these patients.[67,68,69]

The effect of pituitary adrenal hormones on experimental gastroduodenal ulceration has also been studied. No evidence has been found that these substances have any influence on the rate of epithelial proliferation itself,[70,71] nor has any adverse effect been found in ulcers secondary to the Mann-Williamson operation.[72,73] Similarly no adverse effect was noted on the course of cinchophen ulcer and on gastric ulcer induced by pancreatin[74] although there was thought to be some retardation of healing of the duodenal ulcers which developed following pancreatin but without any increase in the depth of these ulcers. Other investigators have found that the size and depth of penetration of experimental peptic ulcers were increased by the intramuscular administration of ACTH and cortisone.[75,76] A high incidence of re-ulceration was also noted in ulcers that had been previously permitted to heal,[77] thereby showing a hormonal effect on gastric mucosa apparently unrelated to any effect corticosteroids might have in interfering with normal repair processes.

Acid pepsin versus mucosal resistance. It is apparent that many of the most recent studies have emphasized the role of mucosal resistance to autodigestion in marked contrast with much of the literature in the past several decades, which had laid stress on hydrochloric acid and pepsin and which would sometimes have us believe that hypersecretion alone is of major etiological importance in ulcer disease. Yet it has long been recognized that a locus minoris resistentiae must be present before peptic ulceration may take place. It is well recognized that acid levels are not invariably high in gastroduodenal ulcer. Normal levels, low levels, and indeed achlorhydria by the usual laboratory tests have been recorded in patients with gastric ulcer. Low acid levels are also sometimes found in duodenal ulcer. Several series have been reported where from 14 to 18% of patients with benign gastric ulceration have achlorhydria. And Palmer[13] has reported three patients with gastric ulcer who had pernicious anemia. Even in duodenal ulcer where hypersecretion can often be demonstrated it is quite possible that acid pepsin in itself is not of primary importance because, as Levin, Kirsner and Palmer have shown,[77] the 12-hour nocturnal secretion will be the same in patients with duodenal ulcer disease regardless of the state of activity of the ulcer on either clinical or radiographic examination. A few years ago Martin, Beal, and Glenn[79] revised the old Mann-Williamson operation by transplanting a segment of duodenum together with pancreatic and biliary ducts to the colon or terminal ileum. In these experiments they found that typical peptic ulceration could be consistently developed in that portion of the duodenum which had been left in situ adjacent to the pylorus. On the other hand, in those animals where the transplanted duodenal segments were anastomosed anywhere between 75 and 290 cm. proximal to the ileocecal valve not a single animal was found to develop a duodenal ulcer with survival of these animals up to a period of 2 years. Furthermore, they found that, even in those with a more distal transplantation, if bile salts and pancreatin were given, a longer time was required in order for ulceration to develop. The results of this particular study are reminiscent of the many procedures involving external and internal biliary and pancreatic fistulae carried out 20 and 30 years ago. In summarizing many of these experiments Ivy, Grossman, and Bachrach[1] had concluded that an impairment in the defensive mechanisms was the chief cause of ulceration and that although hydrochloric acid might be essential in peptic ulceration it was therefore not necessarily the primary cause. In another recent significant experiment Cooper, et al.,[80] perfused the esophagi of animals, some of which had been pretreated with adrenal steroids, with gastric juice from other animals and found that the steroid-treated animals were particularly susceptible to esophageal ulceration. They felt that their experiments proved that adrenal steroids diminished resistance of the esophageal mucosa to acid pepsin.

Protective actions of gastrointestinal mucosa. For more than 100 years people have wondered why the stomach does not digest itself. The famous surgeon John Hunter was impressed by the occasional presence of post-mortem digestion of the stomach, and he commented in 1772,[81] "It immediately struck me that it was the process of digestion going on after death, and that the stomach being dead was no longer capable of resisting the powers of the menstruum which itself had formed for the digestion of food." Claude Bernard in 1856 had postulated the presence of a protective epithelium which by a continuous process of desquamation and renewal protected the integrity of the gastroduodenal mucosa, and Harley in 1860 theorized that the gastric epithelium is protected from autodigestion by a protective layer of gastric mucus. The methods by which the gastric mucosa was believed to protect the stomach from digestion have often been reviewed,[1,2] and they

are thought to include the following: (1) the presence of mucus on the surface epithelial cells, (2) the resistance to peptic digestion by inhibition of peptic activity, (3) the capacity of the gastric mucus to neutralize and buffer acids, (4) the tendency of gastric mucus to become more viscous with increase in acidity,[5] the tendency of mucus to cover the gastric mucosa by more or less continuous film. Hollander in recent years has advocated the combination of theories of both Bernard and Harley and has entitled this the two-component mucus barrier.[82] In the past accurate evaluation of the status of mucus secretion by the mucosa of the upper digestive tract has been prevented by the lack of a dependable method to quantitate accurately the amount of mucus secretion that has taken place. Considerable research is proceeding in this field at the present time, and newer methods of chemical analysis such as electrophoresis[83] are currently in use.

Mucin — a sulfur-containing polysaccharide. Much, of course, has been known about gastric mucin for a good many years, and some of this information may well be of the greatest significance. For example, in 1887 Liebermann[84] had found that gastric mucin could be divided into 2 main groups, those that contained sulfur and those that did not. In 1913 Lopez-Suarez[85] and later Levene[86] reported the isolation of mucoitin sulfuric acid from the pig's gastric mucosa. Fifteen years later mucoitin sulfuric acid was isolated from the gastric mucosa of the rat, and the antipeptic activities of gastric mucin were found to be due to this constituent mucoitin sulfuric acid.[87,88,89] At this time the use of powdered hog mucin for the treatment of peptic ulcer was advocated and was currently being given a clinical trial.[90,91] Mucin was also used in this period for treatment of experimental peptic ulcer in animals as was chondroitin sulfate, which was known to be chemically similar in composition.[89,92,93] It has since been found that many other sulfur-containing mucopolysaccharides will inhibit the proteolytic action of pepsin. Such substances as heparin, chondroitin sulfate, Paritol-C[94] and carrageenin[95,96] all have this activity. These and other miscellaneous facts such as the importance of sulfur-containing compounds in nutrition, in normal liver function, and in wound healing, together with several observations that the adrenal steroids might in some way influence sulfur metabolism suggested an avenue for clinical investigation. In 1951, therefore, a program was initiated to determine the effectiveness of methionine in the treatment of peptic ulcer. The basic hypothesis for this study, together with pertinent references to the literature, were included in 2 papers published in 1955[97] and 1957.[98] Over a period of 4 and one-half years 54 patients with chronic peptic ulcer were treated with from 3 to 6 grams per day of DL-methionine, and good clinical results were obtained in 80% of the cases. Although this clinical study was concluded in 1956, several patients actually insisted on continuing treatment, and it may be of interest that one patient took supplemental methionine over a period of 8 years without any evidence of ulcer recurrence. Additional facts of interest have since been found in reference to gastric mucin. Grossberg, Komarov, and Shay[99,100] stated their belief that mucoitin sulfuric acid was secreted by the neck cells of the gastric glands in the body of the stomach. They did not feel that it was secreted in the antral region. Belanger,[101,102] however, by autoradiographic techniques showed that sulfomucoprotein was secreted by the cardiac and pyloric glands as well as by the mucous neck cells in the glands of the body of the stomach. He also showed the presence of this substance in glandular epithelium. Brunner glands of the duodenum did not show evidence of sulfur fixation, and his photographs demonstrated that whereas the glands in the pyloric end of the stomach incorporated radiosulfur this was terminated abruptly at the duodenal side of the pylorus. Jennings and co-workers[103,104] found that the distribution of radioactivity following the administration of methionine-S^{35} was even more diffuse than that following the administration of $Na_2S^{35}O_4$. After methionine, radioactivity could be found in the surface epithelium and also in gastric mucin. Whereas cortisone was found not to have any apparent effect on the incorporation of methionine-S^{35} into duodenal mucus secretion, appreciably less $S^{35}O_4$ was secreted, particularly during the first 2 hours after administration. The chemical nature of mucoitin sulfuric acid has been extensively studied, and it is found to be a polysaccharide consisting of acetylglucosamine, glucuronic acid and esterified sulfuric acid in equimolar proportions in combination with protein.[105,106,107] The electrophoretic mobility of this substance is very closely similar to that of chondroitin sulfuric acid of ground substance. In chondroitin sulfuric acid there is a chondrosamine instead of a glucosamine.

The effects of adrenocortical steroids on mucopolysaccharides. Following the original observation by Ragan and co-workers on the inhibition of wound healing and formation of granulation tissue by the pituitary-adrenal hormones, a large body of confirmatory evidence has been accumulated. The catabolic action of adrenal cortical steroids is well known. Kinsell in 1950 observed the excretion of large quantities of sulfur-containing compounds after the administration of corticotropin or the adrenal steroids.[108] This has been confirmed in animals by Gundersen and Touchstone.[109] Some of the sulfur excreted has been found to be in the form of taurine.[110,111] The inhibition of sulfhydryl metabolism was also observed following the administration of adrenal steroids.[112] In 1951 Layton showed that cortisone would inhibit the synthesis of chondroitin sulfate.[113] It has also been shown that mast cell activity[114] and the incorporation of S^{35}-labeled sulfate into the sulfomucopolysaccharide of mast cells[115] are inhibited by cortisone. Much of the voluminous literature on the

effects of corticosteroids on connective-tissue ground-substance, cartilage, healing of fractures and wounds, and allied subjects has recently been reviewed.[116] The mucopolysaccharides of the gastrointestinal tract were not, however, discussed in this paper.

Hirschowitz in 1955[48] had noted a progressive decrease in gastric juice viscosity after ACTH and cortisone within a few hours after administration. Kammerer and co-workers noted the same phenomenon.[5] Denko has shown that cortisone would inhibit the incorporation of radioactive sulfur into gastric mucin,[117] and Halme and co-workers[118] found that cortisone acetate reduced the synthesis of glucuronic acid of the duodenal mucosa by 50%, despite the observation that hepatic conjugation was not similarly affected. They wondered if the synthesis of glucuronides and the secretion of mucoprotein were competitive functions, and they observed that a glucurogenic substance like cincophen might lead to a depletion of mucus secretion and that therefore cortisone and cincophen might both be ulcerogenic for similar reasons. Menguy and Masters[119] more recently have found that the intramuscular administration of cortisone to dogs prepared with denervated gastric pouches caused a substantial decrease in the rate of mucus secretion by these pouches. They also found that the steroid induced changes in the quality of gastric mucus, particularly a decrease in the sialic-acid cencentration in the mucin. The terminal position of the sialic acid on the mucin molecule was believed to be largely responsible for those physicochemical properties which account for the protective function of this glycoprotein.

Steroid ulceration in laboratory animals. In 1945 Ingle[120] and co-workers had made the incidental observation that gastric ulcers were occasionally found in rats which had been given daily doses of 5 mg. of 17 hydroxy-corticosterone. In 1951 with cortisone acetate[121] a high incidence of gastric ulceration was observed. At this time they also demonstrated that the incidence and severity of various pathological changes were related to the dosage of the cortisone. These observations were confirmed in 1952 by Baker.[122] In 1953 Welbourn and Code[123] found that corticosteroids had no demonstrable effect on the frequency of forestomach ulcers in the Shay rat. A high incidence of acute erosions in the glandular stomach were once again observed. They also found that corticosteroids had an inhibitory effect on acid secretion. In 1958 Robert and Nezamis found that whereas corticosteroids after 10 to 15 days of treatment in the nonfasting rat would cause ulcers in the glandular stomach, in a high percentage of fasting animals similar ulcerations were found to develop after as few as 4 days of treatment.[124] Kowalewski[125] later found that anabolic steroids would markedly diminish the frequency of steroid ulceration in the rat, although the survival of these rats was not prolonged. He also observed that the radio-activity of the sulfated mucopolysaccharides was reduced in the cortisone-treated animal and that the anabolic hormone Nilevar would prevent this change. Ulcers in the glandular stomach have also been incidentally found in rats treated with the antimetabolite ethionine.[126]

Analysis of recent literature on steroid ulcerogenesis. This then is a relatively brief review of only some of the experimental work which has accumulated in reference to steroid ulcerogenesis. In recent years less emphasis has been placed on the relationship of hypersecretion of acid and pepsin to the etiology of these lesions and more emphasis has been placed on the resistance of gastroduodenal mucosa to autodigestion. Extensive evidence has developed which indicates that the steroid hormones have deleterious effects on the formation of sulfur-containing mucosubstances secreted by the mucous neck cells of the gastric glands and also on those present in the surface epithelial cells. There is also extensive evidence that the adrenal steroids have an inhibitory effect on the synthesis of ground-substance and on related mechanisms of wound healing. Other closely related mechanisms may also be involved. For example, steroid therapy increases the need for ascorbic acid, and it has been found that the formation of ground-substance is interfered with and the uptake of S^{35} is markedly diminished in scorbutic animals.[127] The increased need for pantothenic acid and other B vitamins as well as sufficient dietary protein during steroid therapy has also become obvious. Corticosteroids are catabolic as well as antianabolic, and some of the nutritional depletions found following severe illnesses and injuries are thought to be secondary to adrenal activity and the high tissue levels of adrenal steroids found at these times. There now seems to be no question that steroid therapy is more deleterious to malnourished and debilitated patients than to well-nourished individuals. Anabolic hormones are sometimes found useful in counteracting this catabolic action. A high-protein diet is also useful. Additional potassium makes up for the increased losses of this mineral and additional calcium has also been found to be useful in preventing osteoporosis. By analogy additional sulfur may make up for increased excretion of this substance. Even in the event antianabolic effects of the adrenal steroids are of greater importance perhaps an excess of such substances in the diet would be useful as it has been found that excess methionine will often counteract the effects of the antimetabolite ethionine.

The adrenocortical steroids and clinical peptic ulcer. The interrelationships between steroid ulceration, so-called stress ulceration, and ordinary clinical peptic ulcer, whether acute or chronic, duodenal, pyloric, or gastric, are still obscure; but it is highly probable that there are common denominators in the pathogenesis of all of these lesions. (Fig. 1). Although it has been pointed out that there is no convincing evidence of adrenal disease in ordinary peptic ulcer, and steroid levels have

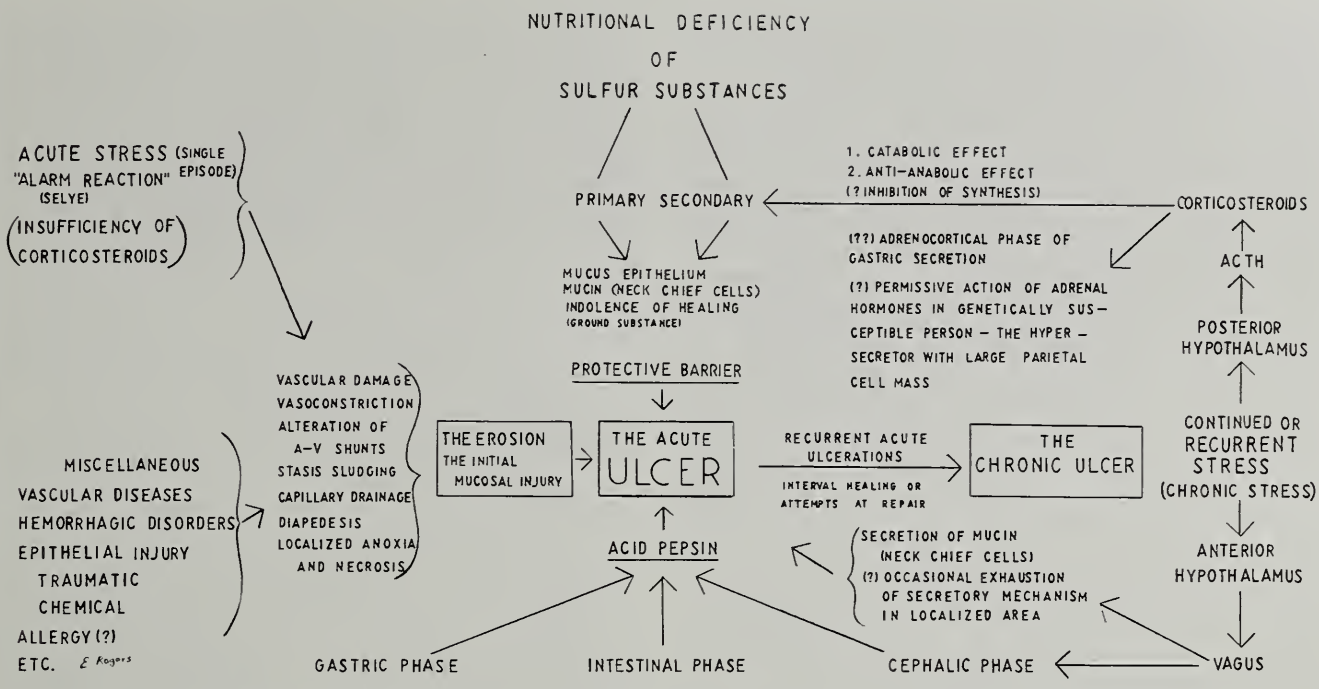


FIG. 1. A hypothetical and schematic chart of the pathogenesis of peptic ulceration demonstrating: (1) the interaction of the acid-pepsin (aggressive) and mucosal (protective) factors necessary for true "peptic" ulceration; (2) the place of a nutritional deficiency of sulfur-containing substances as the most important common denominator in the development of peptic ulceration; (3) the double and paradoxical role of the adrenal cortex in the etiology of many types of ulcers of the stomach and duodenum.

generally been within normal limits, there is no denial of the fact that severe stress, whether due to environmental change, exercise or injury or merely due to emotional reactions,[128,129] is generally associated with adrenal cortical hyperactivity and occasionally high plasma and high urinary excretion levels. It is also probable that all persons are capable of developing gastroduodenal ulcerations under certain conditions of severe stress. Some people may be more prone to such ulceration than others, and this may be partly due to inherited metabolic defects in normal gastric function. The large gastric and duodenal (Curling's) ulcers occasionally seen from several days to a month after severe burns[130] may result from a combination of both catabolic and antianabolic actions. Such ulcerations should, however, probably be differentiated from the small acute erosions seen immediately after the burn injury[131] as these are similar to the lesions described by Cioffi and others after adrenalectomy and attributed by Selye to the alarm reaction or shock phase of the adaptation syndrome. Severe liver disease may also play a significant role in stress ulceration as the metabolism of sulfur amino acids will be found affected. Poor liver function may also be associated with an inability to inactivate steroidal hormones leading to a high circulating blood level of these substances.

Some other harmful side effects of hypercorticism should also be briefly mentioned, namely certain vascular and hematologic changes such as vascular injury, sludging, thrombosis and cellular diapedesis. It will be recalled that cutaneous purpura and ecchymoses are sometimes seen in Cushing's disease. These changes have been theoretically related to mast-cell damage,[132] and mast cells are believed to produce a number of substances including histamine, serotonin, heparin, and sulfur-containing constituents of ground-substance. Histamine[133] has been recently found to induce changes in the sulfate content of the mucosa of the stomach and duodenum.

It was postulated a number of years ago that the adrenal gland may have 2 roles in the pathogenesis of peptic ulcer.[25,28] As the adrenal steroids are part of the defensive mechanisms of the body, important in maintenance of homeostasis and important in regulation of cellular and capillary permeability as well as various enzyme systems, an adequate supply of these steroids might protect the gastroduodenal mucosa from the development of the acute hemorrhagic erosions and the tiny acute ulcers sometimes seen immediately after stressful events. On the other hand it was thought that due to recurrent stress or continuing injury, adrenal cortical hyperactivity would ensue, resulting both in a decrease in defensive properities of the gastroduodenal mucosa and an indolence in the healing of already-established active peptic ulceration, both of these secondary to the catabolic and antianabolic actions of adrenal corticosteroids. In later papers[97,98] the second part of this thesis had been elaborated upon by postulating the presence of a basic nutritional inadequacy of sulfur-containing compounds in peptic ulcer. To quote,

"Such a nutritional deficiency could be primary and due to poor food habits, excessive consumption of highly refined foods or price rationing, or it could be a secondary or 'conditioned' inadequacy[134] and the result either of an interference with digestion, absorption or utilization of essential nutrients, or related to factors which increase their requirements, destruction or excretion. The typical patient with chronic peptic ulcer exhibits a diathesis characterized by gastric hypersecretion and by increased gastric motility, largely due, it is believed, to hyperactivity of the vagus. In this instance we may speculate that because of stress of one kind or another this patient also metabolizes and excretes excessive amounts of sulfur-containing substances such as gastric mucin. (And it is obvious that no cell can secrete continuously.) If because of long-continued stress, the demand then for these substances exceeds the supply, if there is an insufficient supply because of hormonal inhibition, poor liver function, or an insufficient dietary intake due to alcoholism, dietary idiosyncrasies, poverty, etc., then an insufficient supply of these sulfur-containing substances will be present. In various cases of peptic ulcer several of these factors may be operative, and some factors may be alternately of primary importance or only of a contributory importance. For example, in the elderly patient with gastric ulcer not associated with pyloric obstruction or chronic duodenal ulcer disease, vagotonia or the antral factor may not be significant. This type of patient frequently does not show either hypersecretion or hyperacidity. On the other hand, increased mucosal susceptibility would be a factor in the development of the ulceration, and it could well be explained as secondary to one of the various nutritional deficiencies described above."[98]

Conclusions

(1) Twenty-eight cases of steroid ulceration have been described and some of the literature has been reviewed.

(2) A survey of the recent literature on steriod ulcerogenesis and related subjects has been carried out.

(3) Ulcerations first becoming manifest in patients who are receiving therapy with either corticotropin or corticosteroids are believed to be primarily due to the deleterious action of adrenal steroids on gastro duodenal mucosa and on gastric mucin resulting in an increased susceptibility to autodigestion. Indolence in healing due to hormonal inhibition of repair processes is believed to play a secondary role. Poorly nourished individuals are particularly susceptible to this and other harmful side effects of hypercorticism.

(4) The adrenal cortical hormones are believed to play both a preventive role and a causative role in the pathegenesis of many types of gastroduodenal ulceration, and a diagram has been offered showing the relationship of the adrenal cortex to the ulcer problem (Fig. 1).

(5) A major common denominator in the pathogenesis of ulcerations of the stomach or duodenum, single or multiple, acute or chronic, idiopathic or secondary either to "stress" or to steroid therapy is believed to be a primary or secondary "conditioned" nutritional inadequacy of sulfur-containing compounds important in the maintenance of the defensive mechanisms of the gastroduodenal mucosa against autodigestion.

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Benign Thymoma, Refractory Anemia and Hypogammaglobulinemia—Case Report

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The renewed interest in physiology of the thymus gland has been manifested by reports which relate its altered function to acquired hypogammaglobulinemia[1,2,3] to myasthenia gravis,[4] to aregenerative anemia,[5] to Cushing's syndrome,[6] and to pancytopenia, myocarditis, lupus erythematosus and hypergammaglobulemic purpura.[7] Malignant thymomas are frequently associated with myasthenia gravis which may be improved by removal of the tumor, although sometimes the disease may appear after its excision, as has been noted in this hospital on one occasion. Benign thymomas have been associated with plasma cell aplasia (agammaglobulinemia) and with bone marrow aplasia (aplastic anemia). A less common effect of benign thymomas on the bone marrow has been reported as hypoplasia or aplasia of the erythroblasts and their precursors in the bone marrow.[8] This usually results in a refractory anemia which is normocytic and normochromic, with diminished or absent reticulocytes in the peripheral blood and with a bone marrow which otherwise shows relatively normal myeloid and megakaryocytic activity. In an excellent recent review, Roland[8] has reemphasized this syndrome of benign thymoma and refractory anemia and summarized the known reported cases, which with the two reported by him totaled 43, and observed that a cure or improvement in the anemia occurred predominantly in those patients who had had the thymoma removed.

This case report is prompted not only because of the rarity of this syndrome or the incentive thereby to report its occurrence in a man with a large thymoma of long duration, but rather to document the occurrence of an apparent singular initial response in the anemia to therapy as well as a later failure of response to the same treatment when relapse occurred from discontinuance of therapy. Certain other unusual features of the disease in this patient are emphasized.

Case Report

First Admission: This 67-year-old male (Hosp. No. 218-787) came to the Central Maine General Hospital, 10-9-59, in a comatose state. According to his wife, he had been a hard worker over the years and had even had a good-sized garden during the recent summer until general weakness and shortness of breath had forced him to stop one month previously. He had had no known blood loss or melena and had not mentioned soreness of mouth or tongue but had frequently complained of numbness and tingling of the hands and feet. He had lost considerable weight (? 20 lbs.) in the past two years.

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Past History: According to his referring physician, he had been seen in January, 1945, because of upper abdominal discomfort. He gave a history of some cough but his major symptoms were those of gas and epigastric discomfort, for which he was given symptomatic treatment. When next seen in September 1946, he had had more coughing in episodes lasting for 1-2 weeks with production of much grey-green sputum and this current involvement was treated with penicillin. In March 1949, he complained of having strained his left arm and of having some inconstant pain in his chest and of increased cough. Subsequent chest x-ray films showed a mediastinal mass 20 x 17 cm. in size. (Fig. 1). He was then admitted to the Central Maine General Hospital where, upon open thoracotomy the encapsulated mass was biopsied and initially was described as a teratoma by frozen section technic but later was determined to be a benign thymoma. The decision then was that, with consideration of his age, the obvious large size of the mass, no excision should be attempted. Routine blood checks were normal at that time. In the interim between then and the present admission to the Central Maine General Hospital, he had had numerous episodes of fever with cough and increased sputum, each controlled with antibiotics (usually penicillin or one of the tetracyclines). Three weeks before the present admission he had begun to have edema of his feet and lower legs, and when seen, was found to have atrial fibrillation, congestive heart failure, and a grade I systolic murmur in the pulmonic area, none of which had been earlier observed. Anemia was not then apparent. He was digitalized and given a low-sodium diet with control of symptoms. In the week before this admission, he had rather rapidly become more pale and weak, had not revisited his physician because of discouragement over his downward course, and had lapsed into his comatose state on the morning of the day of admission to his hospital.

His Family History was remarkable only in that his father and mother had lived into their 80's and had died of a shock and cardiac trouble respectively; two siblings had died of pneumonia and uremia.

According to his wife, he had mentioned some postural dizziness for 2-3 years; had complained of some shortness of breath for perhaps 10 years; had had a poor appetite for 2 months; and had had moderate constipation for many years. He had continued to be active to the time of his present illness and had had no recent chills, fever, or respiratory infection.

Physical Examination: On admission, the rectal temperature was 98°, the pulse rate 112 per minute, the deep and regular respirations 34 per minute, and the B.P. 104/60. He was noted to be a thin, elderly, very pale, irrational and restless male, with frequent movements of the extremities and some inconstant twitching of the left arm. The pupils were about 3 mm. each, equal, regular, and reacting to light. The optic fundi showed normal discs and pale retinal fields with tortuosity of arterioles, but no hemorrhages or exudates. There was a suggestively sweet odor to his breath, and his tongue, which appeared to protrude slightly to his right, was poorly papillated and slightly red. The neck veins were distended to the mandibular angles in the prone position. The chest was of somewhat increased antero-posterior diameter, the lung fields were clear and resonant throughout, and the percussible

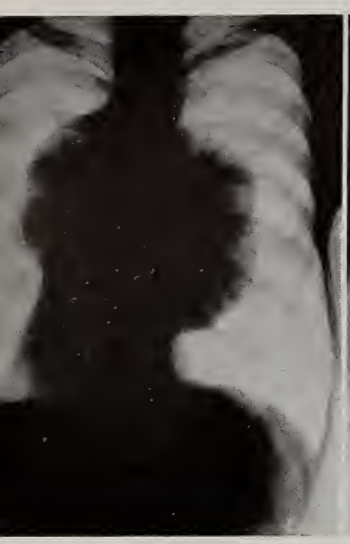


Fig. 1. Roentgenographic film of benign thymoma as first found in 1949.



Fig. 2. Film of chest 10-12-59.



Fig. 3. Repeat film 3-15-60.



Fig. 4. Final film obtained 10-24 shortly before death when clinical pulmonary edema was present.

left border of cardiac dullness was at the left anterior axillary line. A grade II (of VI) blowing pansystolic murmur was best heard at the apex but was nearly as marked over the entire precordium. The abdomen was scaphoid with a rounded non-tender liver edge felt 3 cm. below the right costal margin and the splenic tip was easily palpable 4 cm. below the left costal margin. The prostate was somewhat enlarged and smooth. There was pitting edema of the feet and lower legs. No adenopathy was found. The deep tendon reflexes were hypoactive-to-absent and the plantar reflexes were indefinite due to withdrawal and general restlessness. The cranial nerves were intact except for a questionable deviation of the tongue.

The laboratory findings were: Hemoglobin 1.6 gm. (10%); red blood cells 570,000/cu.mm.; white blood cells 8200/cu.mm.; with 62% segmented neutrophiles, 31% lymphocytes, 6% monocytes, and 1% eosinophiles. The peripheral blood smear showed marked anisocytosis and macrocytosis of the erythrocytes which appeared normochromic. One nucleated red cell was seen and the platelets appeared normal. The reticulocytes were 1/1500 erythrocytes. The urine was yellow, cloudy and acid with a specific gravity of 1.015; 5 mg. per cent albumin but no sugar or acetone was found, and the sediment showed 0-1 white cells, occasional hyaline casts, and frequent uric acid crystals. The BUN was 73 mg. % (later, 15 mg. %). Three stools were negative for occult blood on quaiac testing. No free hydrochloric acid was found in the gastric aspirate. Bone marrow smears showed normal myeloid and megakaryocytic cells, few erythroblasts, no normoblasts and no plasma cells.

Initially, while the laboratory procedures were being accomplished, he was placed on the danger list and given 500 mcg. vitamin B12 subcutaneously. Some 16 hours after admission, he was found in shock with barely perceptible pulse and blood pressure of 70/0. The skin was cold and sweaty. He was given a liter of dextrose in water intravenously and two units of whole blood, following which the blood pressure was 98/40. He was also given Cortef® 25 mgs. intramuscularly four times a day. By the following morning, he was more alert although still somewhat confused, irrational, and uncooperative. A soft rectal impaction was relieved. An x-ray film of the chest showed slight enlargement of the thymoma (Fig. 2). There was no reticulocytosis until 10-21-59 when a 3.6% reticulocyte response was noted. The hemoglobin was 3.5 gms. as a result of four blood transfusions during the period. At this time because of the red cells appearing more hypochromic, iron in the form of Fermalox® was given three times a day orally. By 10-26-59, the hemoglobin was 4.2 gms. with reticulocytes of 6.9% and on 10-28-59 the reticulocytes were 15.1% with hemoglobin 4.4 gms. Subsequent reticulocytosis was 14.7, 10-30-59 and 14.3, 11-1-59. The hemoglobin continued to rise to 9.7 gms. on 11-10-59. By this time, he was much stronger, up and about, eating well, and wishing to go home. He was discharged 11-11-59 to continue taking iron, vitamins, oral corticosteroids in the form of Medrol® 4 mgs. 4 times a day, and to get injections of B12 every two weeks.

Second Admission: He was readmitted to the Central Maine General Hospital 3-14-60 complaining of weakness, dyspnea, palpitation, anorexia, and some weight loss. He had had no chills or fever and there was no known blood loss. For the past 10 weeks, he had had no medications. Until that time, he had continued to take Medrol, iron, vitamins, and had received the B12 injections as prescribed. During the week prior to admission, he had begun to have throbbing, pulsating headaches, and frequently felt a swishing of the pulse in his ears and was quite light headed. His temperature, pulse, and respirations were normal. The blood pressure was 120/52. The tongue was now smooth and pale. The neck veins were markedly distended and there was the same cardiac enlargement to the left with a grade II pansystolic apical and precordial murmur. The liver edge was felt 3 cm. below the right costal margin and the spleen was easily palpable 5 cm. below the left costal margin. Both legs were edematous. No vibratory sensation was found in either lower leg to the hips.

The hemoglobin was 2.4 gms.% and the red cell count was 1.0 million/cmm. The white cell count was 6300 with 65% segmented neutrophiles leukocytes, 1% stabs, 32% lymphocytes, and 2% monocytes. The red cells were of varying size and shape, but appeared normochromic. The urinalysis was negative. The BUN was 25 mgs.%. The first reticulocyte count was 0.1%; thereafter, none were seen at any time. The stools were guaiac negative. The bone marrow was again hypoplastic, particularly with regard to red cells. An x-ray film of the chest (Fig. 3) showed enlargement of the mediastinal mass. The serum proteins were 6.3 gms.% with albumin 3.7 and globulins 2.6 gms.%. A routine paper electrophoresis of serum proteins revealed the gammaglobulin concentration to be only 0.5 gm.%. Except for some elevation of alpha-2 globulins, the other serum protein components were normal. During the next two weeks, he was transfused several times and oral iron therapy was continued. There was no reticulocyte response to this and administration of B12, 1000 mcg. a day subcutaneously, was started. There was no reticulocyte response to this within 10



Fig. 5. Non-traumatic rupture of spleen, 625 gms. Immediate cause of death from lethal intraabdominal hemorrhage.

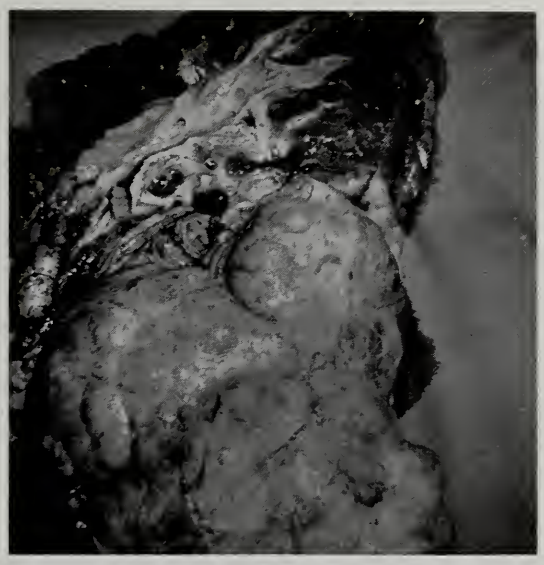


Fig. 6. Extremely firm encapsulated and lobulated mediastinal tumor, 19 cms. diameter, weighing 2050 gms. more or less completely distorting or compressing all of the great vessels at hilum of heart and lungs.

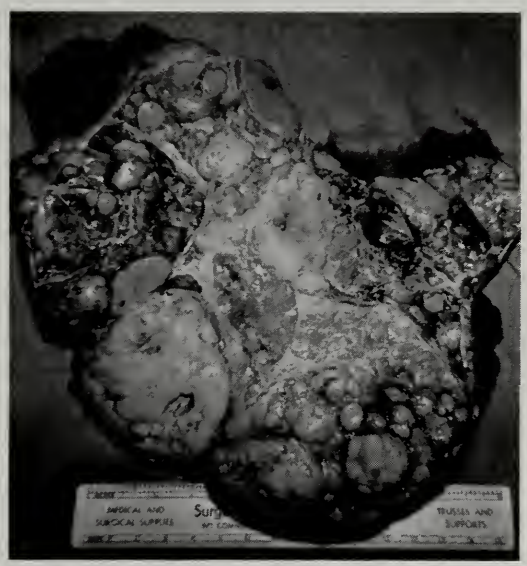


Fig. 7. Cross section of tumor showing irregularly lobulated and trabeculated pattern, with minimal stroma, poor vascularity and focal necrosis.

days, and Medrol 4 mgs. four times a day was added to the regimen. Again, there was no reticulocyte response, but he was feeling better, probably as a result of the multiple transfusions so that he was discharged again taking the same medications as on the previous admission.

In the interim between this last discharge and his last admission, he was seen weekly for repeat transfusions (40 in all) and intramuscular injections of 100 mgs. of testosterone propionate. At no time was there found any reticulocyte response and from May to October 1960 no reticulocytes were ever found. During this time, the WBC dropped to increasingly lower levels and the blood platelets became markedly diminished as estimated from blood smears.

Final Admission, 10-2-60: This occurred because of the sudden onset of lower anterior substernal chest and upper epigastric pain, dyspnea, shock state, and diffuse pulmonary rales of two hours duration. He was dyspneic and orthopneic, very pale, and perspiring profusely. Many petechial hemorrhages of the skin were visible. The legs were again edematous. There was marked venous distention and apparent cardiac enlargement as on previous occasions with many inspiratory crepitant-to-coarse rales throughout both lung fields with expiratory wheezing. The hepatosplenomegaly was still present. He seemed to respond to repeat transfusions, nasal oxygen, and diuretics. A few hours after admission, he collapsed on attempting to get out of bed, and no pulse or blood pressure was obtainable. He responded to two additional transfusions of blood and following this there was marked abdominal distention and spasm with diffuse tenderness. His hemoglobin was 6.4 gms. with hematocrit of 16% and the next day the hemoglobin was 4 gms. with hematocrit of 10.5%. The total white cell count was 800 and these were mostly lymphocytes. No platelets were seen on smear. Two stools gave a 4+ guaiac test. The urinalysis was negative. The BUN was 34 mgs.%. Two blood cultures were negative. X-ray films of the chest showed continued enlargement of the thymoma and pulmonary edema (Fig. 4). On the morning of the third hospital day, the previously mentioned abdominal findings increased suddenly in intensity and more petechiae of the trunk and legs were noted. He quickly became more dyspneic and pale and died two hours after onset of the increased abdominal symptoms.

On autopsy, two hours after death, the skin showed many areas of purpura and petechial hemorrhages. Upon opening the abdomen, there was found approximately 3 liters of bloody fluid. This was seen to have come from a 10 cm. tear in the spleen on its lateral midaxillary surface. The spleen (Fig. 5) was enlarged with weight 525 gms. and showed one area of extravasated blood clot in the lower tip. The liver of weight 1525 gms. showed gross evidence of chronic passive congestion and hemosiderosis. The stomach was atrophic as were the adrenals which weighed 10 gms. The pancreas and kidneys were not remarkable. In the chest, there was a very large mediastinal mass which had displaced the heart downward and outward (Fig. 6). Although encapsulated and somewhat lobular, it was dissected free only with difficulty due to an adhesive pleuritis. Its weight was 2050 gms. The cut surface was a homogeneous greyish-white with lobulations and trabeculations (Fig. 7). The lungs were atelectatic and there was mild bronchiectasis of both lower lobes. The aortic arch had also been pulled downward along with the heart. The latter weighed 300 gms. and the myocardium was pale and flabby.

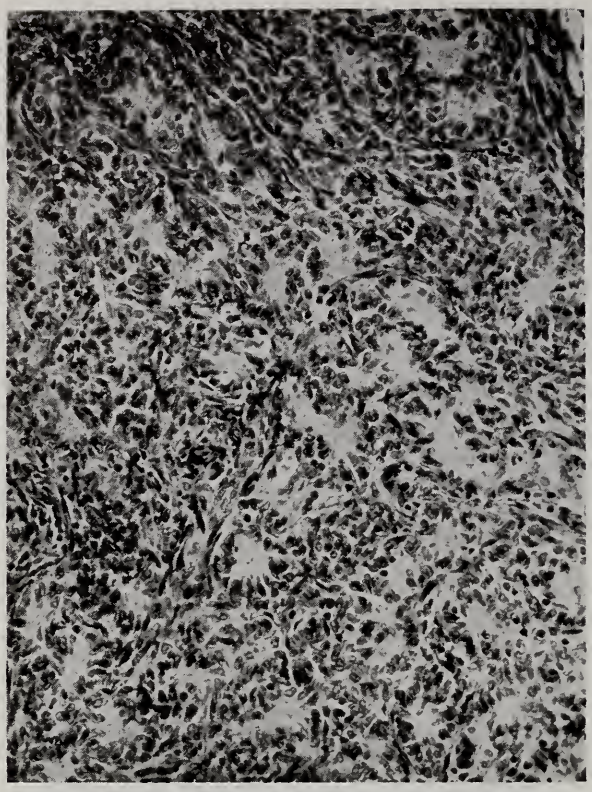


Fig. 8. Typical field from cellular area in tumor, showing epithelial cells arranged in rosettes, pseudoacini or perithelial cords. No mitotic activity. Poorly vascular and scant stroma. X 300 H and E.

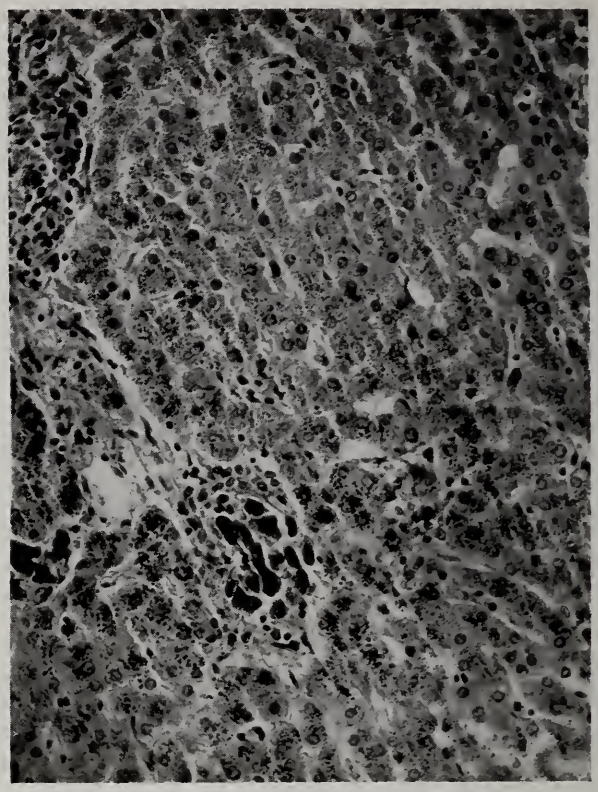


Fig. 9. Section of liver, showing intense hemosiderosis of parenchymatous and Kupffer cells about portal canals. X 300 H and E. Iron stain positive.

There was moderate atherosclerosis of the coronary vessels and aorta. The bone marrow was extremely pale.

Microscopically, the thymoma was found to consist of spindle-shaped clear epithelial cells in some areas with many lymphocytes in other areas (Fig. 8). Bone marrow sections showed practically no cells except lymphocytes. Sections of the liver (Fig. 9) showed hemosiderosis present.

Discussion

In summary, this 69-year-old male who for 12 years had been known to have a large mediastinal mass proven to be a benign thymoma, first presented with a severe anemia and responded to transfusions and a combination of hematopoietic stimulants. He then did well for four months. When he voluntarily discontinued these drugs, he quickly relapsed and the recurrent severe anemia was refractory to the same treatment regimen which had previously been successful. During the last few weeks of life, there was gradual development of severe leukopenia and thrombocytopenia. Death occurred as a result of the latter with hemorrhage into the spleen, rupture of the latter, and massive intraperitoneal hemorrhage.

The thymoma was undoubtedly present for many years prior to its discovery by chest x-ray films in 1949, as judged from its slow increase in size which was observed during the last eleven years. It was a large mass occupying 1/3 of the thoracic cage. In spite of this, there was a surprising paucity of local symptoms occurring as a result of its presence. There had been no chest pain, pleurisy, hemoptysis, dysphagia, or laryngeal nerve involvement, and until the onset of the anemia, he had been able to work without particular shortness of breath or orthopnea. The tumor probably caused, or at least promoted, the increased tendency to episodes of bronchial infection and recurrent pneumonitis. When first discovered and biopsy tissue was obtained, the diagnosis of a benign thymoma was made. Because of its position and size, the lack of symptoms, and evident benign structure, and in view of his age, its surgical removal was not attempted or advised.

With exception of recurrent pulmonary infections, he then remained well until onset of congestive heart failure and anemia. These had been of rather rapid onset some two weeks prior to his first hospital admission and his last illness. Marked pallor, atrial fibrillation, and a harsh precordial systolic murmur were then noted for the first time. He responded somewhat to digitalis administration but soon had to be hospitalized because of onset of shock symptoms and coma. The onset of the anemia had been so rapid that his physician noted he must have lost blood in some way. However, there was no history of either hemorrhage or bleeding. Because of the suggestive history of numbness and tingling in hands and feet, the appearance of the tongue and the evident macrocytosis of red blood cells when the blood smear was initially examined, the first impression was that he might have pernicious anemia and that this had probably been present for some time. How-

ever, as his course unfolded, there was evidence that the anemia was not related to deficiency of either B12 or of iron and was not related to hemolysis. The relationship of the anemia to the thymoma was not, therefore, considered. However, it was not until terminally that pancytopenia was evident.

In tracing the series of events from the time of his first hospitalization, it seems probable that some mechanism was suppressing the bone marrow and that this was at first correctable, and later was not. Roland[8] summarized the possible course of refractory anemia associated with benign thymoma and emphasized that the theory most tenable, namely, that the tumor produces some bone marrow depressive substance, has not been proven in repeated attempts. An auto-immune concept, although attractive in view of the occasional associated hypo- or agamma-globulinemia, has likewise not been proven. In any event, as Roland concludes from his review of the 43 reported cases to the present time, while the relationship between benign thymoma and refractory anemia is unknown, the association is at least well documented. The anemia is usually severe and associated with a high mortality. Of all medications and therapy, including thymectomy, steroids, splenectomy, and androgens, as well as the usual iron, vitamins, B12, peridoxine, and folic acid, only two patients had remissions who had not had either removal or irradiation of the thymus. Eight of 22 patients who had had thymectomy had remissions. All but one had had prior irradiation or removal of the thymoma, however, all other forms of treatment were uniformly ineffective. Of the thymomas which were reviewed, there was an association of the anemia with spindle-type cells of the tumor in about 70%.

Thus, the disease in the patient described in this report conforms to the concepts of this syndrome in that a refractory anemia was associated with a large benign thymoma. Unlike those reported previously, however, the anemia did initially respond to corticosteroids, vitamin B12, and iron with a satisfactory reticulocyte response. This remission lasted at least 3 months and relapse occurred only after the therapy had been voluntarily discontinued. A subsequent similar attempt with the same therapy was unavailing. It seems possible that he could have had a longer life had he continued the treatment regimen.

Jacox, et al.[9] have recently described an adult male patient who, two years after surgical removal of a large thymoma, was found to have a significant reduction of plasma$_1$ A$_1$ gamma$_1$ M and gamma$_2$ globulins associated with a marked reduction of plasma cells in bone marrow and lymphatic tissues. In their review of this and of other reported cases which they were able to find in the medical literature, they emphasize the association of thymic tumor, hypogammaglobulinemia and absence of plasma cells in lymph nodes and bone marrow. Their patient had leukopenia, failed to mobilize granulocytes when challenged with Pyrexal and developed no antibodies with repeated injections of tetanus toxin and typhoid vaccine. A cytomegalovirus was repeatedly isolated from his sputum and tissues.

This same association was evidenced in the patient herein described. It seems possible that his repeated pulmonary infections may have been related to the hypogammaglobulinemia.

Summary

1. An instance of acute anemia associated with a large benign thymoma known to have been present for more than 12 years is reported. Hypogammaglobulinemia was also present.

2. This anemia initially responded to transfusions, corticosteroids, and hematopoietic stimulants with a sustained reticulocyte response and rise in hemoglobin.

3. This remission lasted until the patient discontinued all therapy. A similar treatment of the relapse which quickly occurred was completely unavailing. Complete bone marrow aplasia was the final result.

4. The syndrome of benign thymoma, refractory anemia, and hypogammaglobulinemia is briefly reviewed.

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237 Turner Street, Auburn, Maine

Maine Heart Association Notes

Effectiveness of an Intensive Coronary Care Area

"An intensive coronary care area is the ideal location for the treatment of patients suffering from acute myocardial infarction. Electronic equipment can be used with maximal effectiveness. Constant, well trained nursing service . . . contributes materially to his recovery. The coronary unit leads to a higher type of medical care and to more frequent visits by the attending physician. The mortality rate . . . can thus be reduced. The incidence of successful resuscitation, . . . when unexpected cardiac arrest occurs, is much higher than on the general floors of the hospital.

"One hundred and twenty-six cases of acute myocardial infarction have been treated . . . with an over all mortality rate of 15.9%. In 16 patients unexpected cardiac arrest developed and 9 of these patients were discharged from the hospital alive and well. Electronic monitoring with a combined automatic external pacemaker is of great value, especially in the acute coronary patient who has varying degrees of A-V block. . . . Five days appears to be the optimal monitoring time since 95% of all expected deaths and unexpected arrests occurred within this period."

Reference: Day, H. W. The American Journal of Cardiology, Volume 15, pages 51-54, 1965.
Submitted by Jacob B. Dana, M.D.

A Statement on the Prevention of Bacterial Endocarditis

The American Heart Association Committee on Prevention of Rheumatic Fever and Bacterial Endocarditis, this month, has released a revised statement on patient therapy.

The statement, in leaflet form, recommends that patients with rheumatic or congenital heart disease who will undergo certain types of dental and surgical procedures should receive antibiotics. Suggested treatment schedules for the protection of these patients are outlined.

The first section of the statement deals with dental manipulation, oral surgery, and bronchoscopy. For these procedures penicillin is the drug of choice. The second section is concerned with theinstrumentation of gento-urinary tract, surgery of the lower intestinal tract, and childbirth. With these procedures a penicillin regimen in combination with streptomycin is recommended. Copies of the statement can be obtained, free of charge, by writing the Maine Heart Association.

Important Days . . .

. . . to mark on your calendar



The 112th Annual Session

Maine Medical Association

The Samoset, Rockland, Maine

The Program

for this Annual Session includes . . . .

SCIENTIFIC SESSION SPEAKERS

- Levine of Brookline, Massachusetts on "Some Clues in the Care of Cardiacs"
- Crile of Cleveland, Ohio on "The Role of Simple Mastectomy in the Treatment of Cancer of the Breast"
- Fremont-Smith of Brookline, Massachusetts on "The Medical Treatment of Rheumatoid Arthritis"
- Gallagher of Boston, Massachusetts, Bean of Owl's Head, Maine and others — Panel — "Your Children and Mine — Their Problems From 12 to 20"
- Bowman of Pownal, Maine on "Psychiatry and Criminal Responsibility"
- Dubord of Waterville, Maine on "The Attorney General's Office and the Investigation of Crimes in Maine"

AFTER DINNER SPEAKERS

The Reverend T. S. Honan, an authority on art, literature and philosophy, of Brewer, Maine on "Medicine and Religion"

Joseph M. Hayman, Jr., M.D., Dean of Tufts University School of Medicine of Boston, Massachusetts on "Trends in Medical Education"

Watch your mail for . . . .

PROGRAM DETAILS

SPECIALTY GROUP PROGRAMS

PROGRAM FOR THE LADIES

RESERVATION FORMS

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

The Effect of Temperature on Cancer

PITTSBURGH, PA. — University of Pittsburgh scientists have found that a cancer of the rat responds sensitively to changes in temperature.

Because the responses to temperature include some of the changes wrought by x-rays and anti-cancer drugs, the research raises the question of a possible role of temperature in cancer treatment.

The American Cancer Society reported these results in research by Drs. Joseph Leighton, Leonard Merkow, and Gerald Justh of the Department of Pathology. The society helps support the research.

The scientists experimented with the highly malignant Walker Carcinosarcoma, which grows under the skin of the rat as a large mass, or in the abdomen as an ascites tumor. After cutting a window in the egg shell, they injected a few drops of the suspension of cancer cells on a membrane of the chick embryo. A large tumor mass developed within a week after inoculation. Immediately after injection the eggs were divided into three groups. One was incubated at the normal temperature, 100° F., another at a cool temperature of 90° F., the third at a warm temperature 108° F.

At the normal temperature the tumor grew as a carcinoma. . . . At the cool temperature the tumor grew as a sarcoma. . . . At the warm temperature the tumor was carcinoma-like, and contained many giant cells. Such giant cells are sometimes seen in human tumors after x-ray and anti-cancer drugs.

The cancer tissue from the embryos at both the normal and cool temperatures, when injected into rats, produced large tumors that were undifferentiated.

The tumors from the warm embryos grew very poorly when injected into rats. The fact that two different kinds of cancer — carcinoma and sarcoma — arose from the same original tumor at different temperatures may indicate that the growth of sarcoma might be partially discouraged by warmth and that of carcinoma might be depressed by cold temperatures.

In addition, in certain types of cancer, the induction of fever in combination with either x-ray or anti-cancer drugs might increase the effectiveness of the treatment.

From American Cancer Society News Service Release

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Medical Care in Public Assistance During the Fiscal Year 1964

EDSON K. LABRACK, M.P.H.*

Expenditures for medical care through State and local public welfare programs in Maine amounted to nearly $8 million during the 12 month period ended June 30, 1964. This was nearly 25% of all public welfare expenditures during the period. About $6 million of this money was spent to provide medical care under State public assistance programs of Aid to Aged, Blind, and Disabled; Aid to Dependent Children; and Medical Aid to Aged. This report concerns these recipients of medical care.

Data in the report represents medical care actually received during the fiscal year, hence moneys shown in this report will vary slightly from financial statistics which represent bills paid during the fiscal year. Data are obtained from hospital discharge reports, nursing home bills, clinic bills, and other bills for medical services provided for public assistance recipients.

Trends In Medical Care Programs

Detailed statistics on medical care were first available for the fiscal year ended June 30, 1962, the year during which MAA program was initiated in the State. During the following years, expenditures for medical care programs, particularly MAA, have expanded substantially. Total expenditures were up by 45.3% from $4.1 million in fiscal 1962 to $6.0 million in fiscal 1964. In MAA program total expenditures rose by over 200%, from $0.4 million in 1962 to $1.3 million in 1964. Expenditures for other programs increased by 26.0%.

In MAA the main reason for the increase was growth of the program, initiated in the second quarter of fiscal 1962, to a full blown program. However, on all programs there were increases both in utilization and in average costs of medical care. This is reflected by increases in recipient rates, number of hospitalizations, duration of hospitalizations, mean per diem expenditure for hospitalization, number of patients in nursing homes, and mean duration of nursing home stay.

*Director, Division of Research and Vital Records

TABLE 1

Number of Recipients of Medical Care and Total Recipients by Program.

Program	*Total Recipients*	*Recipients of Medical Care*
AABD		
Aged	12,900	4,658
Blind	440	102
Disabled	2,800	815
AFDC		
Adults	7,300	1,103
Children	20,500	1,013
MAA	3,610	3,610

TABLE 2

Medical Care Recipient Rates per 1,000 for Persons Receiving Public Assistance: Maine, Fiscal Years 1962, 1963 and 1964.

Program	*Recipient Rates* 1964	1963	1962
AABD			
Aged	361.1	365.9	335.2
Blind	231.8	288.9	215.2
Disabled	291.1	320.7	290.2
AFDC			
Adults	151.1	160.4	167.5
Children	49.6	58.7	55.4

Recipients of Medical Care

During the fiscal year there were approximately 47,500 persons in the State who received public assistance. These included aged, blind and disabled persons in AFDC families and recipients of MAA. Of these persons 11,301 or 18.8% received medical care: 8,946 were hospitalized, 2,922 received nursing home care, and 210 received other services including visits to comprehensive medical clinics, prescribed drugs, and other care. Table 1 shows recipients for various programs.

TABLE 3

NUMBER OF RECIPIENTS OF HOSPITAL IN-PATIENT CARE AMONG PERSONS RECEIVING PUBLIC ASSISTANCE IN 1964 AND RECIPIENT RATES FOR 1962, 1963 AND 1964, BY PROGRAM.

Program	Recipients	Recipient Rates		
	1964	1964	1963	1962
AABD				
Aged	2,623	203.3	216.8	196.4
Blind	80	181.8	160.0	163.0
Disabled	573	204.6	207.5	196.2
AFDC				
Adults	1,102	151.0	160.1	166.4
Children	1,013	49.4	58.7	55.4
MAA	3,555			

TABLE 4

TOTAL HOSPITAL DAYS AND AVERAGE DURATION OF HOSPITAL EPISODES FOR PUBLIC ASSISTANCE RECIPIENTS 1962, 1963 AND 1964.

Program	Total Days	Average Days per Hospitalization		
	1964	1964	1963	1962
AABD				
Aged	48,391	13.5	12.7	12.5
Blind	1,065	15.8	14.6	15.7
Disabled	10,225	10.9	8.9	10.7
AFDC				
Adults	9,904	6.6	6.5	6.4
Children	7,952	5.8	5.8	6.0
MAA	74,323	12.6	11.5	10.9

TABLE 5

MEAN EXPENDITURES PER PATIENT HOSPITALIZED AND PER TOTAL PATIENT FOR PUBLIC ASSISTANCE RECIPIENTS, 1962, 1963 AND 1964.

Program	Average Expenditure					
	Per Patient Hospitalized			Per Total Patient		
	1964	1963	1962	1964	1963	1962
AABD						
Aged	$348	$323	$293	$ 71	$ 70	$ 58
Blind	258	227	235	47	36	38
Disabled	329	328	278	67	68	55
AFDC						
Adult	185	172	147	28	28	24
Child	155	125	120	8	7	7
MAA	364	341	275	359	338	275

The recipient rate for medical care among all persons receiving public assistance, exclusive of MAA, was 175.0 per 1,000 in 1964, slightly lower than in 1963, but higher than in 1962. Trends in receipt of medical care during the past 3 years are not clear. Table 2 shows recipient rates during each of the past 3 years.

HOSPITALIZATION

A total of 8,946 public assistance recipients were hospitalized during the fiscal year 1964. The total number of hospital days was 151,860. This was an increase of 9.9% over the number of hospital days in fiscal 1963 and an increase of 59.4% over the number in fiscal 1962. The total expenditure for hospitalization was $2,778,691. This was slightly over $200,000 more than expenditures for hospitalization in fiscal 1963 and $1.1 million more than in fiscal 1962.

Recipient rates for hospitalization were lower in 1964 than in 1963, but were higher than in 1962 except for AFDC recipients. See Table 3.

The number of separate hospital episodes, as measured by the number of hospital discharges of public assistance patients was 12,100 during 1964. Multiple hospitalizations were common. The average number of hospital discharges per recipient of hospital care was 1.5. The average number of hospital days per recipient of hospital care was 17.1. The average duration of a hospital episode was 13.5 days for recipients of all types. The average duration ranged from a high of 15.8 days for MAA recipients to a low of 5.8 days for children in families receiving AFDC. The average duration of hospitalization appears to be increasing on all programs. See Table 4.

The average expenditure for hospitalization was $58 per recipient of public assistance and $311 per recipient hospitalized. The mean expenditure per patient in 1964 was $27 more than in 1963 and $78 more

TABLE 6

NUMBER OF RECIPIENTS, RECIPIENT RATES AND EXPENDITURES FOR NURSING HOME CARE, BY PROGRAM: MAINE, 1964.

Program	Received Care	Recipient Rate	Expenditures		
			Total	Average Per Patient	Average Per PA Recipient
AABD					
Aged	2,593	201.0	$2,824,109	$1,089	$219
Blind	30	68.2	33,318	1,110	78
Disabled	297	106.1	316,301	1,065	113
AFDC					
Adult	2	0.3	296	148	

TABLE 7

NUMBER AND PER CENT OF PUBLIC ASSISTANCE PATIENTS IN NURSING HOMES, BY MONTHS OF CARE: MAINE, 1964.

Months of Care	Number of Patients	Per Cent of Patients
Total	2,918	100.0
Less than 1	235	8.0
1	427	14.6
2	192	6.6
3	184	6.3
4	110	3.8
5	131	4.5
6	97	3.3
7	108	3.7
8	116	4.0
9	96	3.3
10	136	4.7
11	240	8.2
12	846	29.0

than in 1962. Table 5 shows means for the 3 years by program.

Nursing Home Care

There were 2,918 public assistance recipients who were in nursing homes during some part of 1964. There was no change in the number of recipients of nursing home care between 1963 and 1964, but between 1962 and 1963 the number of recipients increased by 21.3%.

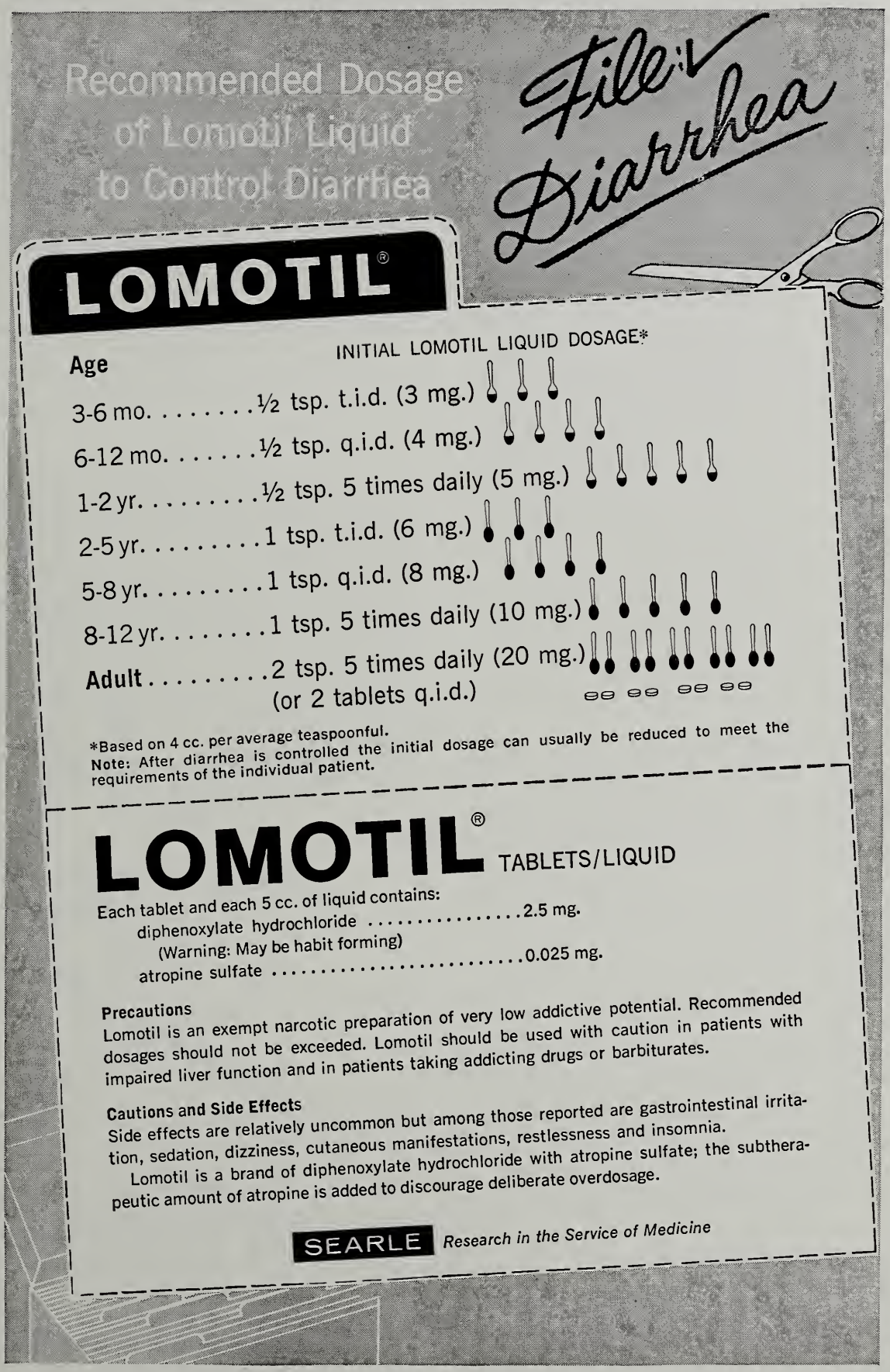
Total expenditures for nursing home care amounted to $3,174,024, nearly $300,000 more than in 1963 and nearly $700,000 more than in 1962. The increase in cost appears to be attributable to an increasing number of long term nursing home patients and a decreasing number of short term patients. In 1964 there were 37.2% of all nursing home patients who were in nursing homes for 11 or 12 months, while in 1963 the comparable percentage was 30.9, and in 1962 it was 27.7. See Table 7.

Other Care

A limited amount of other medical care was provided in the form of clinic care, drugs, and home nursing visits for MAA recipients, and for AFDC recipients selected for rehabilitation. Such care was provided for about 140 patients and the total cost of these services was $11,677.

STEROID ULCERS --- *Continued from Page* 82

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SEARLE Research in the Service of Medicine

County Society Notes

CUMBERLAND

A combined meeting of the Cumberland County Medical Society, the Maine Thoracic Society and the Maine Radiological Society was held on February 18, 1965 at the Holiday Inn in Portland, Maine with 107 members attending.

Following a social hour and dinner, the meeting was called to order by the Vice-President, Charles W. Capron, M.D. who introduced Dr. Alfred Hurwitz who, in turn, presented the speaker of the evening Dr. Felix G. Fleischner, Professor of Radiology, Emeritus, Harvard Medical School.

Dr. Fleischner's topic was Acute and Chronic Pulmonary Emboli and Cor Pulmonale. He stressed that the X-ray appearance of pulmonary infarction is usually not the classical triangular shape described in textbooks. Modern medicine recognizes that the majority of emboli do not go through the full progression to lung necrosis and that X-ray changes may be very transient. Experimental data suggests that debility or other abnormal factors must be present in the body to cause pulmonary infarction by an embolus. Clinical judgment with assistance by X-ray is important in the diagnosis of pulmonary infarction; X-ray must not be depended upon alone. Physicians must consider repeated pulmonary emboli as being a potential cause of chronic shortness of breath.

Dr. Capron announced that late comers, arriving more than fifteen minutes after the appointed dinner hour, would be served only dessert and coffee unless they have called ahead to alert the banquet manager of their late arrival.

Dr. Capron also announced an agreement between the Cumberland County Medical Society, the Portland Medical Club, the Mercy Hospital and Maine Medical Center medical staffs whereby one joint resolutions committee would be appointed by the President of the Cumberland County Medical Society to prepare an appropriate memorium in the event of the death of a member.

A special meeting of the Cumberland County Medical Society was held in the Camden Room of the Eastland Motor Hotel in Portland, Maine at 7:30 P.M. on March 1, 1965.

Alvin A. Morrison, M.D. introduced Niles L. Perkins, Jr., M.D. who presented a resolution which was subsequently adopted by a vote of a majority of the assembled members:

> "Be it resolved: That the Cumberland County Medical Society support the concept of a unified medical complex involving the Maine Medical Center, the Mercy Hospital and the Portland City Hospital."

Dr. Perkins presented further suggestions concerning possible economies and efficiencies to be achieved by unification of acute medical facilities. There was considerable generalized discussion of the matter.

The second definite action voted upon by the members was that Drs. Alvin A. Morrison, Maurice VanLonkhuyzen and Niles L. Perkins, Jr. rewrite the informal elaboration of potential benefits from unification before passing the information on to the Medical Advisory Committee for presentation to the hospitals and agencies involved.

STANLEY B. SYLVESTER, M.D.
Secretary

KENNEBEC

The Kennebec County Medical Association met on February 18, 1965 at the Holiday Inn in Augusta, Maine.

Paul H. Pfeiffer, M.D., Delegate to the American Medical Association, reported on the special meeting of the House of Delegates of the AMA which was held in Chicago, February 6-7, 1965. He explained the position of the AMA backing HR 3727 officially known as "The Eldercare Act of 1965." Frank B. Bull, M.D. made the motion that the Kennebec County Medical Association go on record as endorsing AMA's action in backing HR 3727. A vote of 34 to 2 carried the motion.

Mr. William B. Wadland, trust officer with the National Shawmut Bank of Boston, then addressed the meeting speaking on A Retirement Program for the Physician. He spoke of the Beacon Investing Corporation group rate annuities which are available to physicians and a retirement program as related to the self employed retirement act of 1962. Mr. Wadland introduced Mr. Francis Howe, also a trust officer with the National Shawmut Bank, and Mr. Howe very clearly and with diagrams illustrated a few basic steps in planning an estate. The interest of the Association was witnessed by the number of questions asked from the floor.

EARLE M. DAVIS, M.D.
Secretary

WASHINGTON

A regular meeting of the Washington County and St. Croix Medical Societies was held at the new Down East Community Hospital on March 2, 1965 with nine members and seven guests present.

Mr. Sanfrid Ruohoniemi welcomed the members and their guests to the hospital and conducted them on a tour of the entire facilities of the hospital. This was followed by a luncheon prepared by the Woman's Auxiliary of the Down East Community Hospital.

The two groups held a business meeting, the main topic of discussion being to obtain a pathologist to serve the local hospitals in Washington County and in Charlotte County, New Brunswick.

John Kazutow, M.D. of Columbia Falls was elected to represent Washington County on the Maine Medical Association's Health Insurance Committee.

KARL V. LARSON, M.D.
Secretary

New Members

CUMBERLAND

Alfred E. Darby, Jr., M.D., 295 Longwood Ave., Boston, Massachusetts
George Sirodot, M.D., 62 Coyle St., Portland, Maine

KENNEBEC

Jacques M. Delphin, M.D., Hudson River State Hosp., Poughkeepsie, New York
Paul A. Marshall, M.D., 59 Edwards St., Portland, Maine
Walter Rohm, M.D., Box 724, State Hospital, Augusta, Maine

PENOBSCOT

Joseph S. Brito, M.D., St. Joseph Hospital, Bangor, Maine
Cecil J. Metcalf, M.D., St. Joseph Hospital, Bangor, Maine

Deceased

KENNEBEC

Charles E. G. Shannon, M.D., 9 Park St., Waterville, Maine, February 2, 1965

News, Notes and Announcements

American Cancer Society 1965 Scientific Session

The 1965 scientific session of the American Cancer Society will be held at the Drake Hotel in Philadelphia, Pennsylvania on June 16, 1965.

A symposium on *Hormones and Chemotherapy for Cancer – A Critical Appraisal* is open to all members of the medical and dental professions and students. There is no advance registration or registration fee.

Topics of interest will include: *Problems and Pitfalls in the Evaluation of Chemotherapy,* Dr. David A. Karnofsky; *Chemotherapy of Chorioepithelioma and Related Trophoblastic Diseases,* Dr. Griff T. Ross; *Chemotherapy in Childhood Cancer* (Except Leukemia), Dr. W. W. Sutow and *The Molecular Biology of Development,* Dr. James Bonner.

For further information write to: Director of Professional Education, American Cancer Society, 219 East 42nd Street, New York, New York 10017.

Regional Educational Seminar

A regional educational (leadership training) seminar will be held at the New Prudential Building in Boston, Massachusetts on Saturday, May 1, 1965. The sessions will be held from 9:00 A.M. to 5:00 P.M.

The seminar is sponsored by the American Association of Medical Assistants and will be headed up by Mrs. Edith Jones, AAMA Trustee.

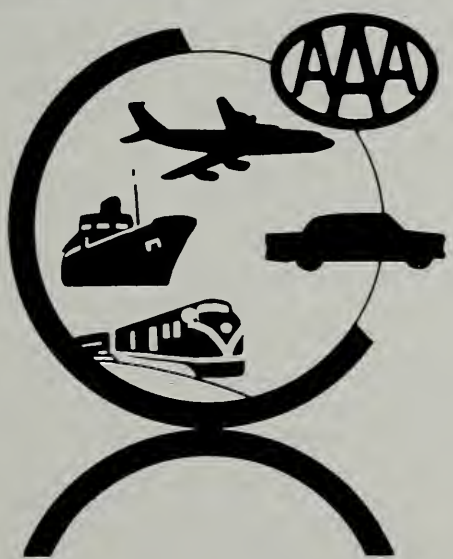
Department of Health and Welfare Division of Maternal and Child Health Including Services for Crippled Children (By Appointment Only)

Orthopedic Clinics

Bangor – Eastern Maine General Hospital
9:00 a.m. and 1:00 p.m.: May 27
Half-day sessions 1:00 p.m.: Apr. 22, Jun. 24
Fort Kent – Peoples Benevolent Hospital
10:00 a.m.: May 12
Lewiston – Central Maine General Hospital
9:00 a.m.: Apr. 16, May 21, Jun. 18
Portland – Maine Medical Center
9:00 a.m.: Apr. 12, May 10, Jun. 14
(In conjunction with MMC)
Presque Isle – Arthur R. Gould Memorial Hospital
9:00 a.m. and 12:30 p.m.: May 11
Rockland – Knox County Hospital
1:30 p.m.: May 20
Rumford – Community Hospital
1:30 p.m.: Jun. 16
Waterville – Thayer Hospital
1:30 p.m.: Jun. 24

Cardiac Clinics

Bangor – Eastern Maine General Hospital
9:00 a.m.: Apr. 9, 23, May 14, 28, Jun. 11, 25
Portland – Maine Medical Center
9:00 a.m.: Every Friday (holidays excepted)



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Cleft Palate Evaluation Clinics

Portland – Maine Medical Center
10:00 a.m.: May 11

Pediatric Clinics

Bangor – Eastern Maine General Hospital
1:30 p.m.: Apr. 23, May 28, Jun. 25
Presque Isle – Arthur R. Gould Memorial Hospital
1:30 p.m.: May 26

Clinics for Mentally Retarded Preschool Children

Waterville – Thayer Hospital
9:00 a.m.: Apr. 7, 21, May 5, 19, Jun. 2, 16
Lewiston – Central Maine General Hospital
9:00 a.m.: Apr. 12, May 10, Jun. 14

Cystic Fibrosis Clinics (In conjunction with the CMGH and MMC)

Lewiston – Central Maine General Hospital
9:00 a.m.: Apr. 2, May 7, Jun. 4
Portland – Maine Medical Center
9:00 a.m.: Apr. 20, May 18, Jun. 15

Dr. Loewenstein Receives Highest Peacetime Decoration

Dr. George W. Loewenstein of Aripeka, Florida received on his 75th birthday the highest civilian peacetime decoration from Germany the "Bundesverdienstkreuz first Class" (Cross of merit), for his lifes work on social legislation and his public health and medical preventive work while in Berlin.

Dr. Loewenstein, a former resident of Chebeague Island, Maine, is a member of the Maine Medical Association and the Knox County Medical Society.

Unanswered TV Questions

"I believe the question related to the overall philosophy regarding Cardiac Surgery at the Maine Medical Center.

"In brief, our goal has been to make available in Maine the most advanced cardiac surgical procedures *of established value and safety,* avoiding the experimental and unproven methods.

"With specific reference to cardiac valve prostheses, our feeling is that these are still in a developmental stage, with many unanswered questions as to valve design and long term results. Accordingly, we have only recently accepted selected patients for possible valve replacement (final decision to be made at time of surgery after direct inspection of valve), when it has become apparent that such patients face early death from advance aortic or mitral valve disease."

HAROLD L. OSHER, M.D.
Portland, Maine

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Book Reviews

Dynamic Pathology, Structural and Functional Mechanisms of Disease. By Maurice M. Black, M.D., Professor of Experimental Pathology, New York Medical College, New York, New York; Attending Pathologist, Flower and Fifth Avenue Hospitals, New York, New York; and Bernard M. Wagner, M.D., Professor and Director of the Department of Pathology, New York Medical College and Flower and Fifth Avenue Hospitals, New York, New York. Approx. 262 pages, 34 illustrations. C. V. Mosby Company, Publishers, St. Louis, Mo.

This very readable book presents in a well organized, almost conversational fashion, the basic newer accepted concepts of normal and abnormal cellular morphology and molecular chemistry which is emerging as the language of the majority of medical investigators today who are worth their N.I.H. grant. Electron microscopes, fluorescent tagging of proteins, photographic visualization of chromosonal abnormalities, a multitude of histochemical procedures and many other popular parameters of scientific study are adding in an explosive fashion newer knowledge of old diseases and unsuspected explanations for other diseases hitherto listed as etiology unknown. As a primer of the newer pathology for the busy but still inquisitive practicing physician whose medical reading and interest take him beyond "Current Therapy" this small, inexpensive text can be recommended.

NELSON P. BLACKBURN, M.D.
Bath, Maine

Synopsis of Pathology. By W. A. D. Anderson, M.D., F.C.A.P., Professor of Pathology, University of Miami School of Medicine; Director of Pathology Laboratories, Jackson Memorial Hospital, Miami, Florida. 6th edition, approx. 875 pages, 406 illustrations, 4 color plates. C. V. Mosby Company, Publishers, St. Louis, Mo.

First published over twenty years ago, this sixth edition synopsis of general pathology presents in the conventional fashion a reasonably comprehensive review of the subject by an author whose background is well established, especially in the teaching field. The book is really an abridged form of the much larger, more definitive textbook of pathology for which the author is editor. The usefulness of this pocket sized soft covered volume, which is well-known to a generation of medical students, is somewhat limited but will undoubtedly continue quite popular with physicians preparing for examinations and the like, and to the increasing numbers of well informed paramedical personnel for general information.

NELSON P. BLACKBURN, M.D.
Bath, Maine

Review of Medical Physiology. William F. Ganong. Lange Medical Publications. $6.50.

Most of us are familiar with this excellent paperback series. The new edition of *Review of Medical Physiology* maintains the usual high standard of clarity. Neat and to the point, the diagrams illustrate the precise and up-to-date text. Highly recommended for anyone about to take a board examination and certainly a worthwhile addition to all medical libraries.

ANTHONY BETTS, M.D.
Portland, Maine

The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, May, 1965 | No. 5

Financing Medical Care Costs

Philip P. Thompson, Jr., M.D.

Introduction

The steady rise in the cost of medical care will continue as it has in the past 20 years. The ability of certain groups to meet these costs is being strained. An increasing number of families are unable to meet them without incurring indebtedness or seeking help from outside the family unit. The burdens of medical expenses are particularly heavy for those families with frequent and/or long-term illnesses and for the aging with reduced incomes.

As physicians, we do too little or are asked for advice too infrequently regarding methods of financing medical care. Most of us have given too little thought to the matter. More frequently, the large medical bill occurs at the time of or shortly after the first encounter with the patient. Patient and public education on insurance and financial matters by the physicians is long overdue.

A greater saving to the patient may be provided by the physicians as a member of his hospital staff or of his county medical society. In this capacity, he can better insure the efficiency of medical services in and out of the hospital. In many larger communities, there is overlapping of expensive facilities, there is reduplication of effort, and poorly organized coordination of hospital and paramedical services.

One of the hopes of reducing the total cost of medical care is to increase the efficiency and organization of those institutions, agencies and persons which provide the medical services.

Of equal importance in reducing costs is the delineation of financial responsibility to the legitimately responsible person or group. With the introduction of a third party between the patient and the hospital or physician, there is less concern on the part of everyone with cost and efficiency. Only as medical groups, labor groups, insurance commissioners are beginning to inquire into utilization of hospital beds, the legitimacy of hospital admissions, the quality of care or questioning or preventing a specific premium increase is there any pressure exerted to curtail the spiral of rising medical costs. The third parties themselves, the employer or the public have not exerted any sustained effort to question, examine or elucidate the problem. There has been too little exchange of information or communication of the facts among these interested parties. Each is playing his own game as he sees his own interest in the immediacy of his own problems.

Each of the facets of this complex social problem will be explored with the benefit of the patient or the public in mind. These facets are to be viewed so the physician may act more intelligently as an advisor to his patient or an advisor to his group which would in turn advise the hospital, the community, or the legislators on problems of medical care financing.

Some of the figures on the cost of medical care have been outlined in a previous paper.[1] Suffice it to say these costs are large. Many people other than physicians and nurses are being employed and paid with this money. Hospital employees, insurance company employees, government workers, builders, and suppliers of food, drugs and medical equipment are among those not generally considered as recipients of a portion of this annual outlay of 80 billion dollars a year for private and public medical care. This is the cost of total medical care for the individual as a private citizen and as a taxpayer. It must be remembered that no matter how it is paid, someone must pay. How can this cost be kept at a minimum without sacrificing quality of the care is the question at hand.

The Individual

The yearly individual out-of-pocket expenditure, ex-

[1]Cost of Medical Care, J. Me. Med. Assoc., April, 1964, p. 60.

cluding his tax contribution, is about 20 billion dollars for personal health services. This includes personal, family and health insurance premium expenses. It represented about 6% of his expendable income in 1963.

The exact amount of his health bill is important as is the amount that he must subtract from his pleasure-producing expenditures, such as recreation, alcohol and tobacco (about 10% of his income). However, his attitude toward this medical expense is even more important than the exact figures. If the individual feels he is getting his money's worth, then the amount he pays for medical care is justified in his own mind. He then, will not resent paying the drug bill, the additional hospital bill, the additional physician charges as well as his insurance premium which he was made to believe would cover it all in the first place. Satisfying the patient by providing top quality and efficient medical care is the sine qua non of any system of providing or financing medical services.

It should be pointed out here that the provision of diagnostic services or unnecessary minor surgical procedures for the "demanding patient" at insurance expense does not usually provide patient satisfaction. This is particularly true when a visit to the psychiatrist or an extra hour of the physician's time would be more rewarding medically as well.

In the consideration of the individual as a payer of medical costs, one cannot neglect the genetic make-up of the patient and his family. There are many patients and their families who can easily be sorted out as those who will require lots of expensive medical care in their lifetime. These are the ones with such illnesses as hypertension, diabetes, psychotic and psychoneurotic traits, and allergies – to mention only a few familial and recurring disorders. This group of patients should be advised to get their insurance early in life, even in their teens, and to get complete preferably "deductible" insurance. They should be advised to set enough money aside to provide for the deductible amount to cover severe or prolonged illnesses.

On the other hand, there are those individuals whose grandparents were long-lived and relatively free of genetic faults which affect health. This group might even be advised to buy no insurance, save a bit for unexpected traumatic or bacterial encounters beyond their control, and take their chances.

The middle group might well be advised to insure themselves early in life against obstetrical and surgical hazards of their early married life and their children's tonsillectomies and appendectomies. During middle age after their youngest is in his teens or out of college, they might well drop their insurance, keep a 'kitty for the unexpected, pay out of pocket for medical bills of a preventative or prophylactic nature. The money they save over a period of years could then go for full medical coverage or deductible type of comprehensive coverage starting at age 60 or 65.

In general, the best method of paying one's expenses for health under ordinary circumstances is to budget and save for "large item" expenses and pay out of the pocket for small ones. This is best not only because it is the cheapest but also it assures one that he is paying only for his own expenses and not for those persons who have higher expenses. This method of financing excludes the administrative costs, which may run 10-50% of the premium, and reduces the "built in" factor of increased utilization of medical care by those who do have insurance. Many other hidden costs are prevented if costs for services do not go through the hands of a third party. Savings of 20-50% of health care costs might be assured by this method, i.e., budgeting, saving, and paying out of pocket.

Prepayment or Installment-Buying Insurance

In general, the types of health insurance are those provided by voluntary health insurance – such as Blue Cross–Blue Shield and private insurance companies. The former are usually primarily service benefits, designed for specific income groups with the ultimate desire of providing "full coverage." The latter are indemnity policies – with set contracted amounts to be paid to the vendor or the patient for specific medical services rendered. Both of these were initially designed to cover high cost items of medical care, such as for hospitalization and surgery. Later, the private insurance companies added another high cost item – long-term illness or "dread disease" contracts. Recently, some companies will insure some individuals against mental illnesses. The policies of private insurance companies are best suited to be tailor-made for peculiar medical potential or to fit the financial and "peace of mind" needs of each individual. If one expects his usage of medical care to exceed the average, he may save money by paying premiums rather than costs. On the other hand, he will be among the "high risk" families who bought the insurance with the same idea.

The "deductible" and "coinsurance" features of many private insurance policies should be explained to patients. The patient should have the "maximum deductible" policy he can afford. This should be the amount he could afford to pay out of his savings and monthly income for medical care. This, of course, would insure the least premium and the most protection.

It should also be pointed out that private insurance companies will insure against other than the usual medical services. Such policies do cover nursing home care, private duty nurses, home nursing care, dental expenses, cost of drugs or just about anything anyone could desire. The premiums for such policies are carefully worked out and are not unreasonable as luxury items go.

There is recent consideration of proposals for Blue Cross–Blue Shield or private insurance companies to act as third party underwriters for government financial programs, such as M.A.A. and other groups whose medical care is provided by The State Department of

Health and Welfare. It makes good sense to have the usual underwriters with their administrative, actuarial and accounting personnel and equipment handle the dispersing of these government funds. The medical profession has the obligation of determining what such a program of medical care should be, and to supervise its operation. This would be a logical extension of the present function of the Maine Medical Association's Health Insurance Committee and/or the Medical Advisory Committee to the State Department of Health and Welfare. These committees are now doing this for Blue Cross–Blue Shield, Medicare, Fedicare, and M.A.A.

Private-voluntary health insurance has demonstrated its capacity and patient acceptance. About 170 million people have this form of insurance. Fifteen million of the eighteen million of those over 65 have some form of voluntary health insurance. By supplementing or augmenting the present policies for those in need, i.e., M.A.A. or other government aided groups, broader coverage could be insured. This added coverage could be partially or totally financed by family, local community, state and federal funds. The delineation of this additional financial responsibility should be an individual consideration. Here is one of the great stumbling blocks to the present extension of coverage. Each group is sitting back and waiting for one of the others to do it or is attempting to pass the buck back and forth to the other.

It might well be pointed out here that of the groups of people not covered by voluntary health insurance most of them either don't need it or don't want it. A large number are already covered by one means or another. Such groups as indigent veterans, retired service personnel, merchant marines, railroad workers, civil service employees and welfare recipients are included in this category. Of those who don't want it, they may fall in that group of unusually healthy persons whose lifetime medical bill might be the single physician house call to pronounce them deceased.

Medical Care Through Taxes

As has been pointed out earlier, three-fourths of the medical care costs, i.e., 60 billion dollars, is already provided by taxes. There has been recently enthusiasm among certain groups to transfer more of the 20 billion dollar individual responsibility to the taxpayer and wage earner.

In this connection, it must be emphasized again that the same costs exist no matter who pays for them and how. Also the fact that the more care provided by a "third party" especially government, the less personal financial concern. As well, the farther away from home the payer, the faster the utilization and total costs will rise. As a corollary, the closer to home an expense is, the more frugal, the more efficient and more legitimate it will be. When the government or industry is paying the bills, the personal interest in economy and efficiency no longer exists. At this point, each individual is interested solely in getting the most for his paid premium or tax. He is not aware of the necessary future increase required by this attitude.

Municipal Taxation for Medical Care

It has been said that ninety cents of each dollar raised by local, city or town taxes are returned to the taxpayer, usually in services. The tax load here falls on the home owner and local industry and business. At this level, it should be pointed out that here one receives his greatest return on the tax dollar. Expenditures are carefully watched by interested citizens. Those with legitimate medical need are easily recognized by neighbors and friends. The local newspapers and public citizens are quick to detect local graft, abuses and unnecessary expenditures. Exposure of such local mishandling of moneys cause personal indignation and ire as well as reaction. When this occurs at a state or national level, the concern, anger and corrective action is ineffective or impossible. The proportion of health care financing at the local level is all too little. The difficulties of administration and the ability of local communities to pass this financial burden along up the government scale have limited the expansion of care at the community level. Certain segments of medical care, such as home care, nursing homes, homemaker programs and other in and out of hospital care should probably be locally financed and administered.

State Taxes for Medical Care

Seventy to eighty cents of the tax dollar raised by the state taxes are returned, usually in the form of services. The tax burden in this instance is distributed more evenly than the municipal revenue. All income brackets are taxed via the sales and other special taxes, such as gasoline and tobacco. One might say that it is a fairer distribution of the tax burden and should more legitimately be expended for medical care of the citizens.

The State of Maine's expenditure for welfare and health services is small. In 1962, this amounted to 33 million dollars of which 15 million dollars was from federal funds. Of this amount, 24 million was for welfare, six million for state institutions, and three million alone for "Health" – of which only a part was for true medical services.

At the state level, it would seem most appropriate to expand health services and particularly medical care. There is an administrative agency with good potential for efficient disposal and supervision of medical care expenditures. It usually has fair to good liaison with the municipal authorities. This could be improved if definite areas of financial responsibility were spelled out. More uniform services and care can be provided for the smaller and poorer towns. Close accounting of expenditures exists. Administrative costs are relatively low compared with federal expenditures for health and welfare programs. The local physician or medical organization has or could have more control or voice in the type of medical program provided. Much could be said in favor

of increasing the expenditures at the state level for health services and medical care.

Nationwide state and local taxes provide for 70% of all government expenditures for health facilities and 90% for direct health services. We, in Maine, are much less than the national average.

Federal Taxation

Less and less of the tax dollar is returned to the individual taxpayer for goods and services. Even in the field of health alone, a larger portion, perhaps 30-50% is used for administrative purposes by the rapidly expanding agencies of the Department of Health and Welfare. This is not necessarily a debit, for the function of the federal government is to organize, plan and administer. Yet the return becomes thinner and thinner as the complexities of social programs increase, the area of federal endeavor widens, and the bureaucracy snowballs itself. More reams of paper are expended, more directives are passed along, more pamphlets and guides printed so the verbiage overwhelms the local administrator. Less action or loss of time for thoughtful solutions of local problems results. Those with the solutions don't have the problems and vice versa.

Possible Solutions or Improvements in the Financing of Medical Care

Changes in the methods of financing and providing medical care must be slow and evolutionary. The present socio-economic-medical complex has developed slowly. The integration of its many facets is difficult and painfully slow in the face of the rapidly changing pressures put upon it. The rapid rise in costs, the growing number of aging, the advances of medical science, the introduction of third party underwriters, the expectation of all peoples of the miracles of modern medicine, the pressures of certain groups for rapid solutions and government intervention – all have complicated the problem and its solution. However, good beginnings have been made. More capable, responsible people in and out of government are thinking, working, planning and acting to try to solve this complicated problem. There are no quick, easy, magic solutions by merely voting extra dollars to do the job. Such a measure might well create havoc, waste, and resentment. It might well set us back years in the good progress that has already begun.

Medical Responsibility

The steady increase in utilization and costs of medical care will continue for an indefinite period. The portion of each person's income spent for health will continue to rise.

It is the responsibility of the individual physician, organized medicine, hospital administrators, hospital trustees, insurance companies and those responsible for ancillary medical services to work as a team to insure maximum efficiency of total medical care. There must be a maximum return on each dollar spent for medical care.

The physician as captain of the medical care team must not only direct and guide his patient through the complexities of the medical, as well as the financial maze. He must also guide his hospital trustees and other groups providing care or dealing with its organization. He has to coordinate the community effort as well as the patient's so each group can unite to a common purpose – better and efficient patient care. Waste of money, manpower, and effort cannot be endured in the face of present and rising costs and medical manpower shortage.

A thorough evaluation of the health of an individual by a careful painstaking history and physical exam is the essence of good medical care. A copy of this recorded examination and all subsequent examinations, medical episodes and laboratory tests should be the permanent property of an individual, or his family and even his descendants. Such a record would prevent unnecessary duplication of physician's time, patient's time and expense. This is particularly applicable since the population is on the move. A variety of physicians are consulted for sundry problems and hospitalization may take place in several hospitals. A form is available called "Medical Passport" and may be obtained by writing: Medical Passport Foundation, 35 E. 69th Street, New York, New York.

After the initial examination, periodic examinations and evaluations should be done. The interval of these evaluations should be a part of the medical advice to the patient. This interval will depend upon age, state of health and history of familial disease.

Hospital Responsibility

Hospital administrators, trustees and the medical executive committees have a heavy responsibility for the cost and efficiency of hospital and total community medical care. Too often each hospital is considering only their own domain with little regard for the community as a whole as an integrated medical care unit. Much duplication of effort and manpower saving could be achieved by the joint meetings of all individual hospital Boards of Trustees and others involved in the total health care of a community.

The complicated business of running a hospital rests in the hands of its Board of Trustees. This group usually consists of local businessmen who are neophytes in the area of medical care. They must depend on trusted medical advisors and administrators. In recent years "hospital consultants" or efficiency experts have arrived on the scene to help in long-range planning in addition to solving immediate problems. The cost of such an expert evaluation and advice runs about $15,000. It would seem that each hospital, if it is to perform at top efficiency, should have this sort of advice at varying intervals. The financing of such an evaluation presents a real problem to the average hospital. The basic cost

should rightly fall on those who are to benefit financially from such an evalution. These might include local citizens and businesses, Blue Cross-Blue Shield, the private insurance industry, United Community Services, State Department of Health and Welfare, voluntary health agencies and even private medical foundations.

An even more vital and cost-saving function of such experts would be the coordination of health services within a city, a region or even several counties. A single consultant for all hospitals with the support of the hospitals, the medical profession, and the communities involved could do much toward the solution of rural medical care and duplication of effort. This might well result in a vast saving in costs of medical care and improved efficiency.

SUMMARY

As the provision and financing of medical care becomes more complex so do its solutions. Only as the united efforts of all concerned, which are well begun, will continue and expand can we hope for a better solution. Good patient-care within his ability to pay for it is the goal. Informed, actively participating and cooperating interested people charged with this responsibility can achieve this goal.

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Carcinoma of the Breast - Some Imponderables

RONALD A. BETTLE, M.D.

During the past 20 years there has been no significant change in the mortality figures for treatment of carcinoma of the breast. It continues to be a disease of unknown cause, treated by a system of trial and error. Some 20 years ago, while a resident at the Maine General Hospital, Portland, Maine, I reviewed the tumor clinic cases of carcinoma of the breast for the previous ten year period. It is interesting to read this paper now and find that there has been very little progress made in the results of the treatment of this disease since then. This is not because of a lack of interest or research. Certainly much more is known of its behaviour. At that time hormone therapy was just beginning to be used and the results of hormone therapy in thousands of cases have been documented. Chemotherapy has also been introduced, but women still die from the disease at about the same rate. There has been little change in the overall mortality figures, or in the overall 5-year survival rate which has remained at about 55%.[1] It is true that we have learned to control the disease to the extent that the individual is more comfortable, but we have not been able to eliminate the malady.

Twenty years ago, attempts were being made to classify the disease so the survival figures would be more meaningful. There are many factors which have been classified, but the two basic characteristics are the extent of the disease (Stage) and the type of tumor growth (Grade of malignancy).[2] The significance of the degree of malignancy and extent of spread is well illustrated by Figs. 1 and 2. Haagensen[3] has championed the value of classification and his system is called the Columbia Clinical Classification, but it ignores grade or degree of malignancy. Two other systems are also in use, based on the extent of the disease. One of these is called the Joint Commission System; Fig. 3 however, a uniform system is still not in use!

There is probably one reason why classification continues to be an imponderable. After all, what is being classified? Is it the extent of the disease, or is it the resistance of the patient to the disease? All of us have seen patients who defy any classification; e.g.,

> A 58-year-old woman had had a simple biopsy excision of a 1 cm. mass of the left breast 7 years previously. It was found to be a scirrhous carcinoma, Grade III. She received no further surgery or treatment. A year previously she was seen complaining of a tender nodule in the left 2nd interspace near the sternum. This was 6 mms. in size and proved to be a metastatic carcinoma, Grade III, probably of breast origin. The breast was completely negative. There were no axillary nodes and the internal mammary chain was also negative. She is still living and well.

This is, of course, a bizarre case but it does not alter the recommendation that a uniform system of classification, based on the degree of malignancy and the extent of known spread would be of value in planning treatment, as well as evaluating treatment response.

But classification is still not the primary problem. Until we know the cause of breast carcinoma, treatment will continue to be unsatisfactory. In the past 20 years we have learned some interesting facts about characteristics of the tumor's growth and its response to various forms of treatment; e.g., there is the 60+-year-old woman with carcinoma of the breast who responds dramatically to hormone manipulation. She gains weight, feels good, and for a time the lesions melt away. Fig. 4

FIG. 1

THE FIVE YEAR SURVIVAL RATES IN THE RESPECTIVE GROUPS AND IN THE GRADES WITHIN THESE GROUPS

Grade	*Stage I*			*Stage II Lymph node involvement*			*Stage III Distant Metastasis*		
	Number of cases	*Five year survival*		*Number of cases*	*Five year survival*		*Number of cases*	*Five year survival*	
I Papillary Adenocarcinoma	2	2	100%	0			0		
II Adenocarcinoma	5	4	80%	4	2	50%	1		
III Scirrhous carcinoma	19	8	42%	19	4	21%	1	1	Simple mastectomy
IV Carcinoma Simplex	1	0		3	1	33%	0		
Total	27	14	51.5%	26	7	25.2%		1	6%
Cases treated by X-ray only							14	No biopsy	
							16		

FIG. 2

THREE YEAR SURVIVAL RATES

Grade	*Stage I*			*Stage II*			*Stage III*	
	Number of cases	*Three year survival*		*Number of cases*	*Three year survival*		*Number of cases*	*Three year survival*
I Papillary Adenocarcinoma	2	2	100%					
II Adenocarcinoma	2	2	100%					
III Scirrhous carcinoma	5	3	60%	4	2	50%	1	0
IV Carcinoma simplex				2	1	50%	1	0
Total	9	7	78%	6	3	50%	2	0

Again, an imponderable – exactly what effect does the manipulation of hormones have on carcinoma of the breast? Twenty years of research has not answered this question. Why should estrogen be effective in producing a regression in advanced carcinoma of the breast in approximately 1/3 of the cases of those 5 years or more past the menopause while castration helps nearly the same percentage pre-menopausally. Then, to further confuse the picture, although Kennedy and Fortney[4] found that castration helped 46% of pre-menopausal women, in young women under 35 castration was not as effective. Then again, those who respond to oophorectomy will subsequently do better with an adrenalectomy or hypophysectomy than those who do not. If manipulation of the corticosteroids fails, we can always try cortisone. The only common denominator seems to be that manipulation of hormones benefits about 1/3 of the patients from the standpoint of comfort and, to some extent, longevity and there seems to be no way to select who will respond – certainly one of the imponderables.

Even more recently, by a system of blind and double blind studies, various chemotherapeutic agents have been added to our armamentarium. The effectiveness of these drugs is now under intensive study coordinated by the National Institute of Health. There is some indication that adjuvant therapy at the time of surgery is beneficial. But the effect does not seem promising for treatment, per se.

The original chemotherapeutic agent of the present series was introduced by Goodman in 1946 – just about 20 years ago. Other than the original agent Nitrogen mustard, the following are found to be effective in the treatment of carcinoma of the breast: Thio-

Fig. 3

Clinical Staging System*

CANCER OF THE BREAST

T	N	M
(Local Tumor)	**(Regional Lymph Nodes)**	**(Distant Metastasis)**
T_1 Tumor of 2 cm. or less in its greatest dimension; Skin not involved, or involved locally with Paget's disease.	N_0 No clinically palpable axillary lymph node(s) (no metastasis suspected).	M_0 No distant metastasis.
T_2 Tumor over 2 cm.; or with skin attachment (dimpling); or nipple retraction (subareolar tumors). No pectoral muscle or chest wall attachment.	N_1 Clinically palpable axillary lymph nodes that are not fixed (metastasis suspected).	M_1 Clinical and radiographic evidence of metastasis except those to homolateral axillary or infraclavicular lymph nodes.
T_3 Tumor of any size with any of the following: skin infiltration, ulceration, peau d'orange, skin edema, pectoral muscle or chest wall attachment.	N_2 Clinically palpable homolateral axillary or infraclavicular lymph node(s) that are fixed to one another or to other structures (metastasis suspected).	

CLINICAL STAGE CLASSIFICATION

STAGE I

No clinically palpable axillary lymph node(s) (no metastasis suspected). (N_0)
Tumor of 2 cm. or less in its greatest dimension (T_1).
Skin not involved (T_1), or involved locally with Paget's disease (T_1).
No distant metastasis (M_0). — $T_1N_0M_0$ or
Tumor over 2 cm. (T_2); or
Skin attachment (dimpling) (T_2); or
Nipple retraction (subareolar tumors) (T_2).
No pectoral muscle or chest wall attachment (T_2).
No distant metastasis (M_0). — $T_2N_0M_0$

STAGE II

Clinically palpable axillary lymph node(s) that are not fixed (metastasis suspected) (N_1);
Tumor of 2 cm. or less in its greatest dimension, as in Stage I (T_1).
Skin not involved, as in Stage I (T_1), or Paget's disease, as in Stage I (T_1).
No distant metastasis (M_0). — $T_1N_1M_0$ or
Tumor over 2 cm. as in Stage I (T_2); or
Skin attached (dimpling); or
Nipple retraction (subareolar tumors), as in Stage I (T_2); or
No pectoral muscle or chest wall attachment, as in Stage I (T_2).
No distant metastasis (M_0). — $T_2N_1M_0$

STAGE III

No clinically palpable lymph (node(s) (no metastasis suspected) (N_0).
Tumor of any size with any of the following associated findings; skin infiltration, ulceration, peau d'orange, skin edema, pectoral muscle or chest wall attachment (T_3).
No distant metastasis (M_0). — $T_3N_0M_0$ or
Clinically palpable axillary lymph nodes(s), that are not fixed (metastasis suspected) (N_1).
Tumor of any size with any of the following associated findings: skin infiltration, ulceration, peau d'orange, skin edema, pectoral muscle or chest wall attachment (T_3).
No distant metastasis (M_0). — $T_3N_1M_0$ or
Clinically palpable homolateral axillary or infraclavicular lymph node(s) fixed to one another or to other structures (metastasis suspected) (N_2).
No distant metastasis (M_0). — $T_3N_2M_0$ or
Any combination of N_2 with M_0. — $T_1N_2M_0$ or $T_2N_2M_0$

STAGE IV

Any stage, with distant metastasis (M_1).

SUMMARY OF STAGE GROUPINGS

STAGE I — $T_1N_0M_0$ or $T_2N_0M_0$
STAGE II — $T_1N_1M_0$, $T_2N_1M_0$ (includes all N_1M_0 except for T_3).
STAGE III — $T_3N_0M_0$, $T_3N_1M_0$, $T_3N_2M_0$, $T_1N_2M_0$, $T_2N_2M_0$ and includes any combination of either T_3 or N_2 with M_0.
STAGE IV — Any clinical stage of disease with distant metastasis (M_1).

**American system developed by The Joint Committee on Cancer Staging and End Results Reporting.*

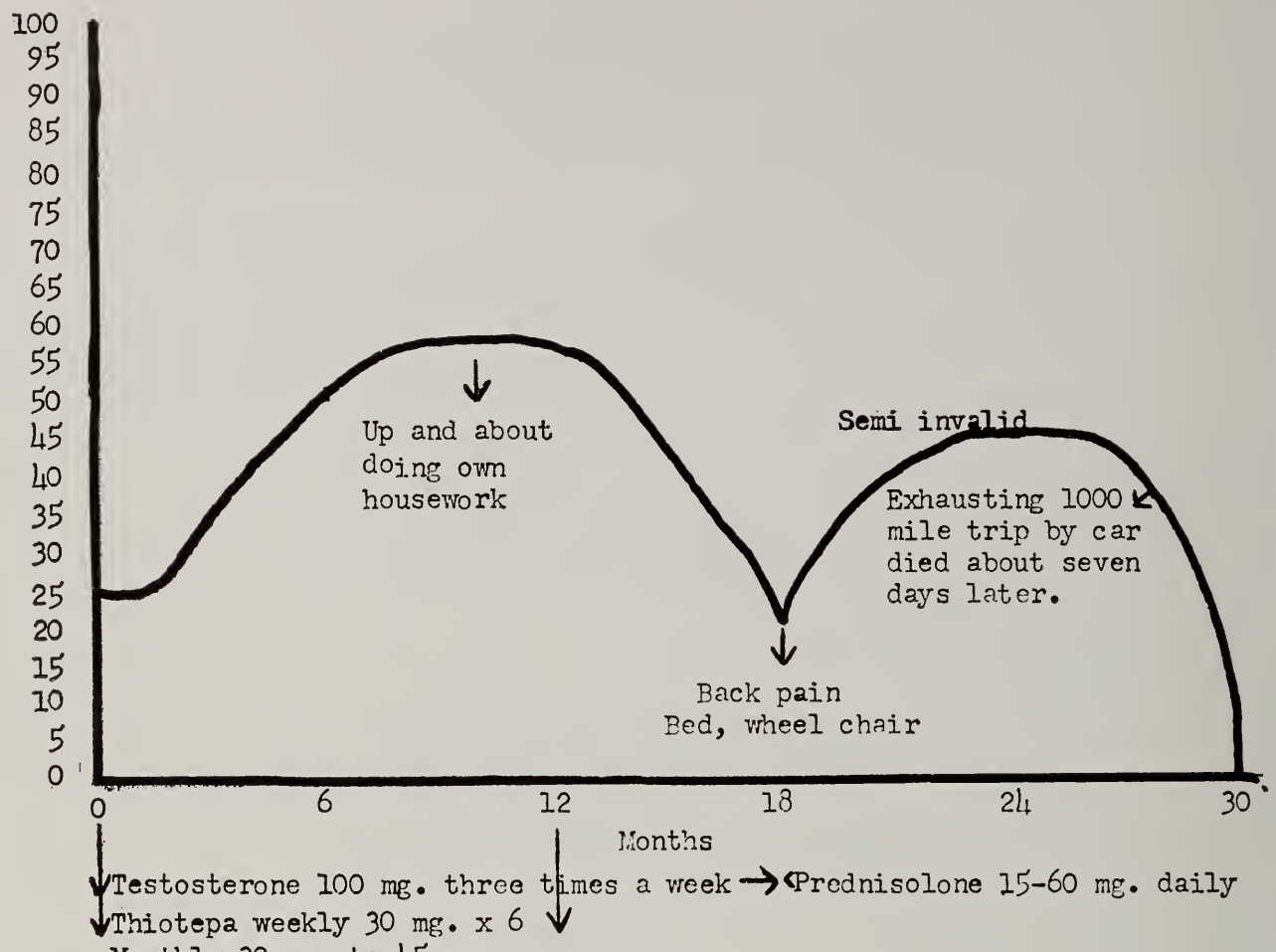


Figure 4 T. O. Age 62 Grade IV Stage III Advanced carcinoma with ulcerations of breast, gross lymph node involvement and x-ray evidence of metastasis to spine, skull & lungs.

tepa, 5-Fluorouracil, Methotrexate, Cytoxan,® Leukeran.® No evaluation of these is to be attempted now, but to add to our imponderable problems – what has been learned about chemotherapy in the last 15 years is again largely by trial and error. One writer has called it "The Russian Roulette System – which one is the live shell?"

Francis Knock,[5] in a recent editorial in S.G. & Obs., Nov. 1964 states, "With the present emphasis on random chemical carcinoma therapy, the wonder is not that so few people have been helped, but rather that any have been helped." He recommends a plan of treatment based on the chemical sensitiveness of the patient's tumor. Dr. Knock believes that cancer cell sensitivity must guide such chemotherapy just as chemical sensitiveness of bacteria now guides antibiotic therapy. This is certainly an ideal to strive for.

This brings us to the most perplexing problem of all. Just what is cancer and how does it get started? This imponderable problem remains basic and some interesting work has been reported by Moore and his group of Roswell Park.[6] Experiments have been conducted in which extracts of malignant human tissue have been injected into mice. The above chart summarizes the results:

	No. of mice surviving 1 yr.	*No. of mice with tumors*	*% of mice with tumors*
1. Malignant tissue	1616	134	8.3%
2. Malignant effusions	446	39	8.8%
3. Benign tumors	76	9	11.8%
4. Normal tissue from cancer free pts.	436	4	0.9%

Approximately 90% of the mouse tumors were mammary adenocarcinomas, regardless of the origin of the cancer site – lung, stomach, etc.

Then there are still the classic experiments with mice by Bittner (1936). These continue to plague one's thinking. What was this tumor factor that was transmitted via milk, blood and semen? After almost 30 years, we still do not have the answer.

We could continue to add other imponderables which have been discovered experimentally and statistically; such as, the effect of heredity, trauma, lactation, x-ray therapy, cystic mastitis, age, enzyme systems, hormone levels, etc. There is also the role of irritation and

coal tar derivatives, etc. Not all of these are factors in breast carcinoma, but they certainly are of considerable interest in the total concept of factors contributing to the development of carcinoma.

It would seem that if we would find a rational therapy for carcinoma of the breast, some hypothesis must be developed and treatment based on this hypothesis. Obviously, this cannot be dogmatic, but would be less frustrating than to continue basing our treatment on statistics, or on trial and error. Suppose we suggest the following hypothesis:

> Breast carcinoma is caused by a tumor factor which alters the growth of epithelium. It begins locally, but once it spreads beyond the breast, even to local lymph nodes, it then becomes a systemic disease. Patients have varying degrees of resistance to the local tumor and even to circulating tumor cells; however, once this resistance is breached, metastatic lesions develop and are the principal cause of death. Treatment should be managed in such a way as to contain, control or eliminate this disease if possible without jeopardizing the patient's natural defenses. Treatment should rather augment the host resistance.

Tumor Factor

We have already discussed the tumor factor briefly and many interesting experiments have been recorded and others are now in progress, trying to determine the specific agent. Since 1876, when Novinsky demonstrated that carcinoma could be transplanted from one dog to another, a search has been made for the causative agent. The most recent agent to be incriminated is the virus! In a recent article published in the *New England Journal of Medicine,* John Enders, Ph.D.[7] summarizes his experience in working in collaboration with Dr. Albert Sabin, using the SV40 virus and the Adenovirus, Type 12 and 18. He states, "The implications of these findings in the future exploration of the possibility that viruses cause cancer in man are obvious and so require little comment." His thesis is that in human cells "the interaction of viruses and cells under conditions such that the cells are not irreparably damaged, but continue to survive and multiply, produce what is called a temperate relation." As pointed out by Dr. Enders, many workers have studied this phenomenon; e.g., "Vogt and Dulbecco showed that malignant changes appeared in isolated normal mammalian cells exposed to oncogenic viruses." As Dr. Robert H. Huebner, Virologist of the National Institute of Health, pointed out, "The question – Do viruses cause cancer? – should be changed to – To what extent do viruses cause cancer?"

A related phenomenon is circulating cancer cells. Warren Cole[8] has written an interesting monograph entitled, "The Dissemination of Cancer Cells." This exhaustive study outlines the extensive work on this problem and demonstrates without a doubt that cancer cells are frequently present in the blood stream of about 50% of patients with malignant tumors. On p. 136 of this monograph is the following comment, which is pertinent:

> "Engell, summarizing the findings of his extensive study, concluded that the histologic grade was probably one of the most important factors in the hematogenous spread of cancer cells. No cancer cells were found in the blood stream in the one case of Grade I differentiation; 33% of the 36 Grade II tumors were positive; 72% of the 36 Grade III tumors were positive, and all of the 3 Grade IV tumors were positive for cancer cells in the blood stream. Engell concluded that cancer cells were probably disseminated from all Grade III and Grade IV carcinomas."

These conclusions corroborate our previous findings relative to the significance of the poorer prognosis in Grade III and IV carcinomas. To add another imponderable-circulating cancer cells have also been found in patients with benign tumors; however, not with the same frequency or implication. Delarue[9] summarized his findings as follows:

> "Only 33% of the patients with malignant disease, who initially yielded one or more positive samples, remain alive and well in a 2-5 yr. follow-up; whereas, 68% of those in whom all blood samples were negative remain free of disease. All patients harboring benign disease are alive and well, regardless of the fact that some showed positive sampling."

Host Resistance

The growth of the tumor seems to progress from breast to local lymph nodes, to regional lymph nodes and throughout the body. Once the tumor has spread beyond the breast it, no doubt, should be considered a systemic disease. But why does it spread? What happens? Is it a breakdown of local or systemic defenses, or both?

Closely related to host resistance is the problem of stress with the resultant depletion of body defenses; e.g., trauma particularly increased the number of takes, as demonstrated by Fisher and Fisher.[10] Clinically, this is manifested in patients after a surgical procedure when, unfortunately, the tumor seems to spread out of control.

An editorial in the J.A.M.A.[11] summarizes it nicely when it states, "that survival or death (from cancer) depends primarily on the imponderables of the tumor-host equation is generally accepted; the importance of host resistance and the ability to reject cancer cells is no longer seriously questioned and may have therapeutic implications."

Treatment

According to our hypothesis, treatment should contain, control or eliminate this disease if possible without jeopardizing the patient's natural defenses. The principal modalities are surgery, chemotherapy, hormones and x-ray therapy. No attempt will be made to discuss the relative value or indications for each type of treatment. This subject is frequently and well written about and the references as listed[3,12,13,14,15,16] indicate some of the current thinking and results, particularly with reference to chemotherapy and hormone therapy.

The following three cases are presented as illustrating various combinations of therapy in advanced cases, with the results. It will be observed that hormone

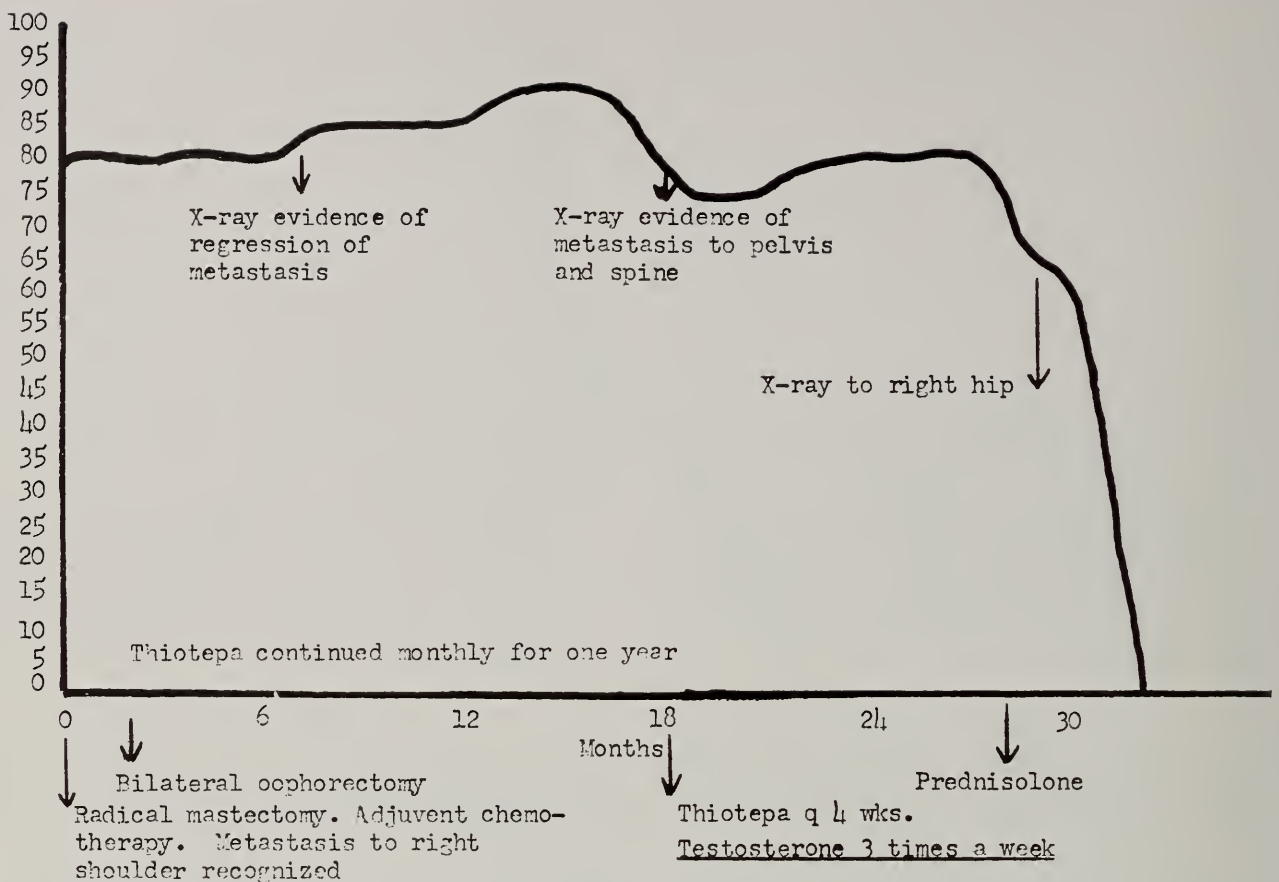


Figure 5 B. L. Age 45 Grade IV Stage III

therapy is usually combined with some other forms of treatment. There is nothing to indicate that hormone manipulation is curative; rather, hormones apparently change the tumor melieu and alter its growth pattern. Combining hormones in metastatic cases with therapy that may destroy or eliminate the malignancy, such as x-ray or chemotherapy, seems more rational.

Illustrative Cases

Fig. 5 — A 45-year-old woman with a lesion 2 x 3 cms., Grade IV, Stage III. Initially the x-rays were read as negative, so a radical mastectomy was done. Within 6 weeks, because of shoulder pain, repeat x-rays were taken and by comparing these with previous films it was recognized that this was actually a Stage III lesion originally.

Fig. 6 — The entire breast was replaced in this 45-year-old woman with carcinoma, Grade IV, Stage II. There was no ulceration but the axillary nodes were grossly involved. First x-rays read as negative. Within 2 mos. bony metastases were obvious and comparison with initial films revealed that early metastasis had been present in the initial films. It should have been a Stage III. Had the bony metastasis been appreciated, this patient would have been treated with oophorectomy and chemotherapy.

Fig. 7 — This 63-year-old maiden lady presented with a lesion classified as Grade IV, Stage III. The entire right breast was ulcerated and satellite nodules covered the area from the clavicle to below the ribs. There were extensive nodes in the right axilla and ulcerating nodes in the right supraclavicular area with extensive edema of the right arm. The entire left breast was firm and the skin edematous with the characteristic peau d'orange. The right 4th rib was apparently involved. Chemotherapy and hormones have kept this patient under control and she is doing her daily office work, and has been for over a year.

Summary — Control vs. Cure

Perhaps we could summarize the results of the past 20 years of treatment of carcinoma of the breast by saying that we have learned how to better control, but not to cure, this disease. Actually, medical therapeutics has many precedents for this concept; e.g., we don't talk about curing tuberculosis, we talk about control. Certainly the term 'Five-year survival rate' in carcinoma of the breast does not give a true picture when the patient is dead in 5½ years from generalized metastasis. But a disease that has been controlled for 5½ years, leaving a comfortable, productive patient the majority of the time, is better than a painful cripple who lives for three years without adequate treatment. Therefore, the management is aimed at using various modalities to achieve elimination of the disease if possible; if not, then containment, comfort and control. This control is based on the hypothesis that the local manifestation is part of a systemic disease due to some, as yet unidentified, tumor factor that responds somewhat erratically to surgery, chemotherapy, hormones and deep x-ray therapy.

For the present we must consider carcinoma of the breast as a disease of unknown cause and must continue to treat it, unfortunately, by a system of trial and error.

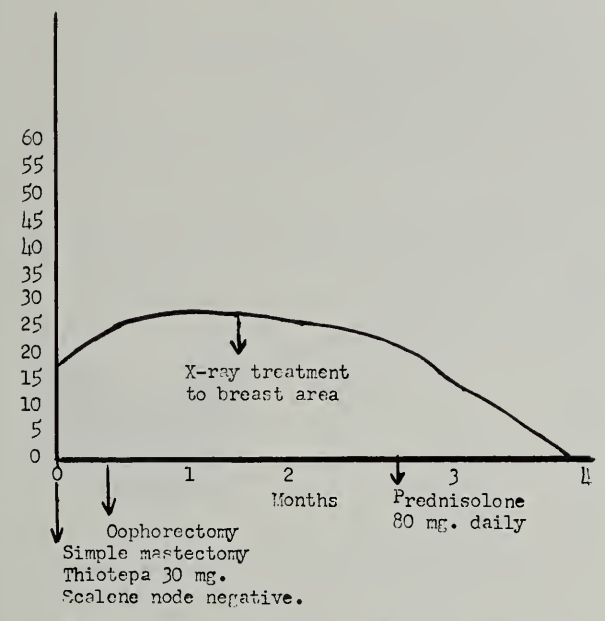


Figure 6 W. R. Age 45 Grade IV Stage III

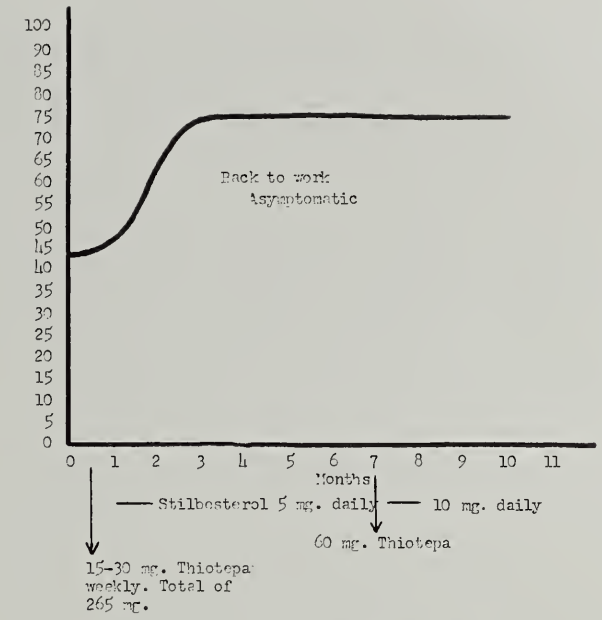


Figure 7 M. M. Age 63 Grade IV Stage III

We can earnestly hope that another 20 years will produce a cure. One thing is certain, and that is, that a uniform system of reporting the Stage and Grade of malignancy would enhance the value of all statistics. As one studies the known facts about this elusive disease, it would seem that the pieces of this puzzle are there if we could just put them together. When we do, each year some 55,000 women in the U. S. will benefit. But, lest we get discouraged, I would remind you of a statement which appeared in the *Annals of Surgery* in 1894, written by no less a surgeon than Halstead.[17]

"We rarely meet a physician or surgeon who can testify to a single instance of positive cure of breast carcinoma . . . Most surgeons operated on breast carcinomas solely for the moral effect on the patient." In that era local recurrences appeared in 60-80% of the cases operated upon. So we have made progress.

Conclusions

1. A uniform system of reporting cases of carcinoma of the breast, based on the degree of malignancy (Grade) and the extent of its spread (Stage) is suggested as important from the standpoint of planning treatment and evaluating the results.

2. A hypothesis upon which to base treatment is suggested, based on accumulating research evidence as to cause. Treatment includes the use of surgery, chemotherapy, hormones and deep x-ray therapy, recognizing at all times the importance of preserving and augmenting host resistance.

3. Cases are presented which illustrate the value of rational treatment, based on classification using the Grade and Stage of the tumors.

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The Detection, Prevention, and Management of Postoperative Pulmonary Complications*

HAROLD D. CROSS, M.D.

Within the past decade thoracic and cardiovascular surgical groups have applied a variety of pulmonary function measurements, including blood gas studies, to their patients in an effort to detect which patients will develop postoperative pulmonary complications. Fortunately, the most valuable screening tests have proved to be as simple as taking a blood pressure. Efforts were then made to prevent these complications by improving lung function. Intermittent positive pressure breathing (IPPB) with Isuprel® (isoproterenol) accomplishes this for the generally recognized complications of pneumonia, bronchitis, and atelectasis. However, there is the acute complication of CO_2 retention which may or may not be predictable, or preventable with IPPB treatments. It occurs in the patient who simply does not ventilate properly because he is infected, obstructed, or physically unable to move his chest. The retention of CO_2 causes an acute fall in pH and the resulting acidosis may be fatal. This complication has not been so routinely recognized and managed because CO_2 retention is *not* acutely reflected in the serum bicarbonate, and the blood pCO_2 and pH which *directly* measure retention and acidosis respectively have not been easily obtained. Modern Technology has altered this situation and the latter two tests are now readily available, and they are fundamental to the diagnosis and management of a complication which may be far more prevalent than hitherto recognized.

Each of the above three areas will be discussed in more detail.

Detection

Concerning detection and the use of simple screening tests, Stein *et. al.*[1] demonstrated that a test of expiratory flow rate was more reliable in detecting which patients would develop postoperative pulmonary complications than routine clinical appraisal, – "for many of the patients in the abnormal group (impaired flow rate) had been passed without concern on the basis of history, physical examination and exercise tolerance (stair climbing)." Their results were Fig. 1:

30 patients with normal breathing test – 1 pulmonary complication.

29 with abnormal breathing test – 20 pulmonary complications.

They employed several tests, but of these the maximal expiratory flow rate (MEFR) correlated best with the complication rate. They used 200 liters/minute as the lower limit for normal for patients over 40 years of age,

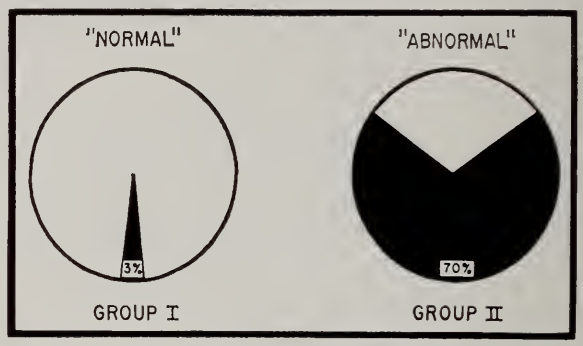


FIG. 1. Reprinted by permission of, Stein *et al.*[1] Percent of pulmonary complications in Group I (normal pulmonary function tests) and Group II (abnormal pulmonary function).

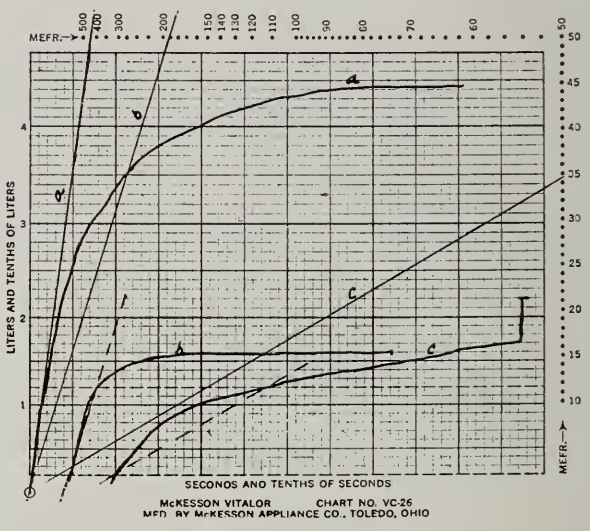


FIG. 2. Graphic Sheet used on Vitalor, from which one can read the vital capacity, maximal expiratory flow rate and FEV_1. Illustrative curves.

		Vital Capacity	*MEFR*
a.	Normal	4.4 Liters	450 Liters
b.	Restrictive pattern	1.6 Liters	190 Liters
c.	Obstructive pattern	2.2 Liters	35 Liters

300 liters/minute when under 40. This relationship between reduced air flow rate and a high incidence of postoperative pulmonary complications raises the question as to how flow rates can be measured. A variety of ways is listed in Table 1. A very simple way is to perform a timed vital capacity, such as is provided by the Vitalor. This test may be performed at the bedside or in the office, taking about three minutes for several readings. The maximal expiratory flow rate (MEFR) is determined by noting the rate of flow of the first liter of expired air after the initial 200 ml. With the grid

*Presented at the Maine Chapter of the American College of Surgeons in Brunswick, Maine on December 9, 1964.

TABLE 1

VENTILATORY TESTS MEASURING FLOW RATES[a,b]

Test	*Normal Range*	*Time Required*	*Apparatus*	*Cost*	*Supplier*
Maximal expiratory flow rate[c,d]	200-500 Liters	1-3 minutes (bedside test)	Vitalor	$150.00	McKesson Appliance Co. Toledo 10, Ohio
		approx. 15 min.	Collins 9 L Respirometer	600.00	W. E. Collins Co. Boston, Mass.
Expiratory Peak Flow rate[e,f]	400-700 Liters	less than 1 min. (bedside test)	Wright Peak Flow Meter	180.00	J. J. Monaghan Co. 500 Alcott St., Denver, Colo.
DeBono Whistle (peak flow)[g]	over 300 Liters	less than 1 min.	DeBono Whistle	5.00	J. J. Monaghan Co.
Maximum Breathing Capacity (MBC)[h,i]	70-130 L. Female 100-160 L. Male	approx. 15 min.	Ventube	85.00	J.H. Emerson Co., 22 Cottage St., Cambridge, Mass.
			Valve, Douglas Bag & Meter	225.00	W. E. Collins Co.
One Second Fast Vital Capacity ($FEV_1\%$)[i]	75% Vital Capacity	3 minutes	Vitalor	150.00	McKesson
			Vitalometer	225.00	W. E. Collins Co.
Match Test[i,j]	extinguish at 6 inches (mouth wide open)	1 min.	Book Matches	nil	

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sheets provided for the Vitalor, direct readings are quickly obtained, see Fig. 2, the vital capacity is also inscribed. Becoming familiar with one simple test of flow rate, rather than struggling with many ways of measuring essentially the same lung function, is suggested.

PREVENTION

Turning to the matter of prevention of pulmonary complications, Anderson *et. al.*[2] treated 42 patients pre- and postoperatively with intermittent positive pressure breathing (IPPB) with Isuprel and demonstrated a marked reduction in the complication rate in those patients with impaired flow (measured by the maximal breathing capacity), Fig. 3. In their untreated control group of 160 patients, there was a postoperative complication rate of 20% contrasted with 2.5% in the treated group of 42 patients. Fig. 4.

Complications were defined as: "temperature elevation, pulse rate elevation, cough or chest pain, or any combination of these accompanied by the radiological demonstration of atelectasis or pneumonitis." Thus one has a choice, prepare all patients with IPPB and Isuprel, or screen them, preparing the patients with abnormal tests.

IPPB and Isuprel treatments presumably resulted in the decreased morbidity by decreasing bronchospasm and improving the flow rate. The effect of bronchodilators on both flow rate and vital capacity is easy to assess and should be determined when either is impaired. The demonstration of its effect on the patient's breathing in graphic form (such as with the Vitalor) may add incentive to the patient and physician that no words may equal.

Having reduced the pulmonary complication rate from roughly 20% to 3% with the above measures, one wonders about the relative merits of such conventional preparations as expectorants, antibiotics, omission of cigarette smoking, postural drainage, chest physiotherapy, the anesthetist's pre- and post-op visits and psychological preparation. These should be continued at least until their essential roles are better defined.

As to the role of the site of surgery in relation to the incidence of postoperative pulmonary complications, Anderson[2] summarized previous reports on this:

2-18% after various G.U. surgery.
3-12% after lower abdominal surgery.
5-42% after upper abdominal surgery.
60% after cardiac surgery.

Anderson's own control group fell into roughly these same percentage groups. He recommends that all patients having abdominal or thoracic surgery be prepared with IPPB and Isuprel.

Now that an approach for detecting which patients

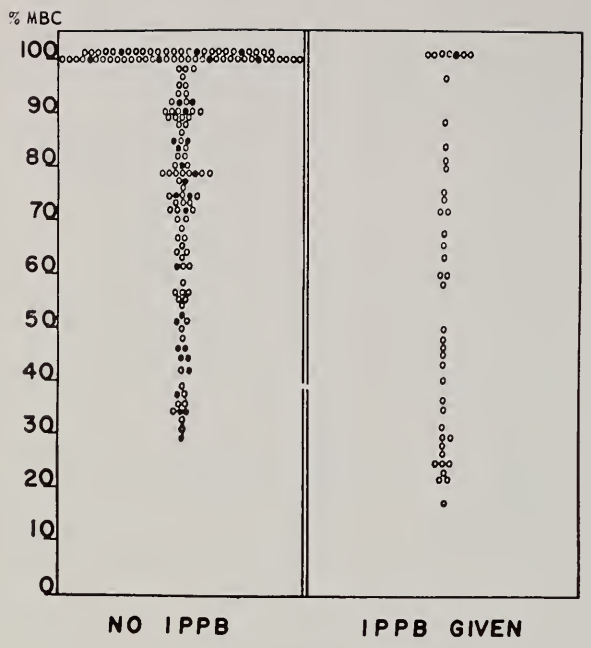


FIG. 3. Reprinted by permission of Anderson *et al.*[2] Maximum breathing capacity as percentage of predicted normal. Black dots represent subjects who had postoperative pulmonary complications, circles represent no complication (pulmonary).

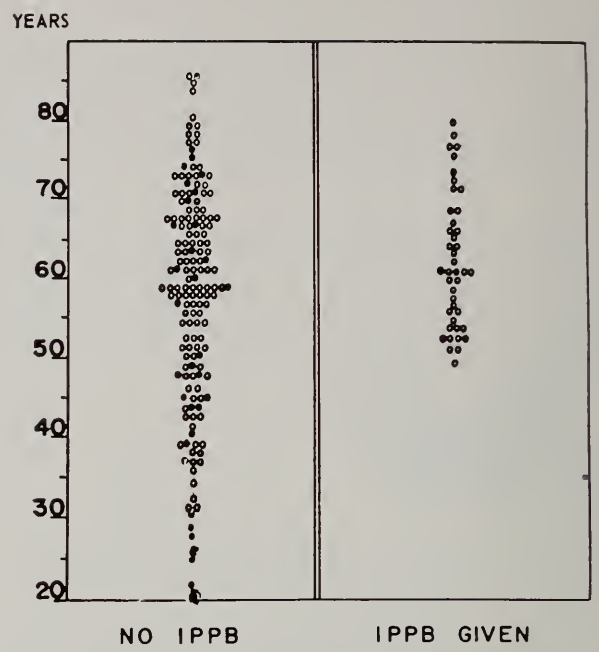


FIG. 4. Reprinted by permission of Anderson *et al.*[2] Black dots represent subjects who had postoperative atelectasis or pneumonia, circles indicate no complication.

are apt to have pulmonary postoperative problems as well as a means of prophyllaxis has been described, there remains the problem of recognizing and treating patients who develop complications.

Complications

Concerning pulmonary complications, there are two general types. The usual type includes pneumonia, bronchitis and atelectasis and these are probably related to an impaired flow rate (the prediction tests) in most instances. Then there is the not so routine type (not because it has not been there but because we have not recognized it) which may or may not be related to chronic and preoperative impaired flow rates and/or lack of IPPB treatments prophyllactically. For example, a patient with perfect lungs can get too much anesthesia, become obstructed or have massive abdominal distension, and thereby develop all the acute pCO_2 and pH problems. He may not be as likely to do this as the patient with impaired lung function, but this may just make it all the more difficult to recognize his problem. One may be alerted to this type of problem by cyanosis, elevated respiratory rate, severe impairment of the flow rate or in the postoperative period (including post-traumatic and post-anesthetic) by any unexplained hypertension, hypotension, lethargy or coma. Having been thus alerted, direct measurement of the blood pCO_2 and pH will provide the answer as to whether adequate ventilation is being achieved. Of the five patients with seemingly minor pre-op elevations of the pCO_2 (over 45 mm Hg, normal being 35-42 mm Hg) in Steins study,[1] all had serious postoperative pulmonary complications. These patients represented the problem of chronic hypoventilation and impaired flow.

Acute respiratory acidosis occurs because of inadequate ventilation in either the patient with normal or abnormal lungs. Its causes include airway obstruction, both upper and lower, depression of the respiratory center from anesthetic and hypnotic agents and inadequate ventilation during anesthesia. This respiratory acidosis or pulmonary failure results in three immediate blood disturbances: rapid rise in CO_2 tension (pressure), rapid fall in pH, and fall in oxygen tension. There is little change in the serum bicarbonate over a several hour period[3] and hypoxia is not invariable as high concentrations of oxygen may be present from a nasal catheter or oxygen tent. Respiratory acidosis can be most accurately and quickly diagnosed by a blood pH.[4] The only cause of sudden acidosis (except from the extreme hypoxia of circulatory arrest) in the postoperative period is hypoventilation, in contrast to metabolic acidosis which requires hours to develop.

To follow a patient with respiratory acidosis under treatment, serial pH and pCO_2 determinations are the best guide posts, not unlike the way the pH is used to manage the patients with diabetic acidosis. For convenience, a dorsal hand vein may be used for the blood sample while the patient is acidotic. This source can be used at other times as long as the hand is warmed to 98°F. and no tourniquet used.[5]

The explanation of the sudden shifts in the pH is that any change in alveolar ventilation such as occurs with airway obstruction, hypoventilation or hyperventilation, will have a prompt effect on the pCO_2 in the

alveoli and blood. Approximately 200 ml. of CO_2 gas are generated per minute in the average sized adult. The only means of excretion is via the lungs. It is thus apparent that any significant and continued reduction in alveolar ventilation will cause rapid accumulation of this waste product. (About 12,000 milliequivalents of CO_2 are excreted by the lungs per 24 hours compared to about 3 milliequivalents by the kidneys.) Because of the direct associated effect on the pH, pCO_2 fluctuations have far reaching implications affecting cardiac, peripheral vascular and central nervous system functions.

That we need the blood pH and pCO_2 for accurate diagnosis and management of a pulmonary failure problem should be apparent from the above discussion. Reluctance to acquire and use this information simply because we "used to get along with a serum bicarbonate," and "used clinical judgement" to determine the other two factors (pCO_2 and pH) is no longer justified. Clinical judgement is not that good,[6] and direct measurement is now easily achieved since the development of reliable pH and pCO_2 electrodes.[7] The mathematics and equations are behind us, so to speak, since no calculations are necessary. All parameters (pH, pCO_2 and CO_2 content) can easily and quickly be measured directly and/or two measured and a nomogram used[8] for the third, Fig. 5.

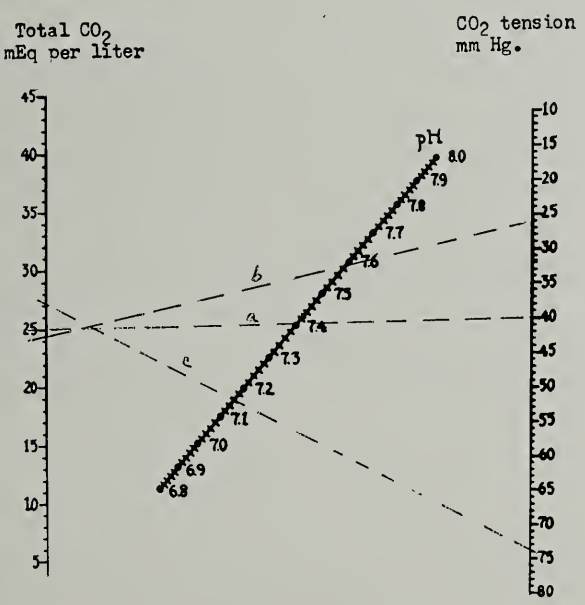


FIG. 5. Nomogram for pH, pCO_2 and CO_2 content. Adapted from ABC of Acid Base Chemistry, The University of Chicago Press, 1955. H. W. Davenport.
a. normal pH, pCO_2 and CO_2 content.
b. hyperventilation for 1 minute.
c. status asthma (hypoventilation).

When the blood pCO_2 is elevated, the lungs are not getting rid of it; if this is acute the pH drops. This process is readily reversible in most instances by adequate ventilation. After a few applications of the pH, pCO_2 and CO_2 content their meanings are grasped, and like other lab data, normal values are best remembered when the tests are used frequently. Work in understanding of these few principles should be more profitable in patient care than wrestling with the technicalities of current literature as to whether the MEFR is better than the MBC, whether the pH and pCO_2 are sufficient, or whether standard bicarbonate, Astrup nomograms, etc, provide clearer pictures of the patient's respiratory status.[9,10,11]

The appendix is a case history illustrating the approach described in this paper.

Case Report

A 71-year-old male was admitted to the G.U. Service on 7/10/64 for prostate surgery because of urinary frequency due to an enlarged prostate. The admission urine showed 20 mg% albumin, 30-40 wbc's, BUN was 17 mg%, IVP normal except for evidence of an enlarged prostate with moderate secondary vesicle changes. He was placed on foley catheter drainage and Chloromycetin®. A TUR was done 7/16/64; the procedure lasted 1 and 3/4 hours; spinal anesthesia was used.

The current pulmonary screening of patients admitted to the ward surgical services includes an expiratory flow rate test. Peak Flow rate was 105 liters (normal over 400), maximal expiratory flow rate 13 liters (normal over 200) and vital capacity of 1.3 liters. There was a history of asthma for more than 40 years, untreated. The patient stated he could walk a mile, slowly. Admission chest x-ray showed diffuse and localized pulmonary emphysema and fibrosis. Because of the abnormal ventilatory studies reported, a medical consultation was obtained. Chest findings were: Respirations 22-24, no wheezes, few crepitant rales at left anterior chest. Because of the marked obstructive pattern found by the two flow tests, the most conservative surgical approach was advised. Blood pH and gas studies were obtained, revealing slight CO_2 retention (48 mmHg., normal 35-42), low pH - 7.32 and near normal PO_2 - 72 mmHg. The predicted CO_2 content was 26 mEq.

The screening ventilation test showed marked impairment of lung mechanics. Because of this finding, the patient was studied for evidence of hypoventilation by blood gas studies. This was confirmed by an elevated pCO_2. A serum bicarbonate would have been normal (26 mEq or less), thus failing to give any indication of this problem. That an elevated pCO_2 should be an alerting sign to postoperative problems was pointed out by Stein[1] in whose study all five patients with pCO_2 over 45 mm Hg had serious complications.

This patient was begun on IPPB with Isuprel one day preop, receiving four treatments, also started on saturated solution of K.I. and continuance of Chloromycetin that had been started for the G.U. infection. The patient tolerated surgery well; IPPB and Isuprel were continued through the early postop period, beginning 2 hours after returning to the ward. His early post-op course was excellent, being out of bed the 1st post-op day. The peak flow was increased to 185 liters on the 2nd post-op day. He remained afebrile and was thought to be ready for discharge approximately one week post-op.

The night of the 8th post-op day, he was noted to be breathless for a short time, relieved by an IPPB treatment. He later became unresponsive, clammy and hypertensive. The resident and intern felt he probably had had a CVA or myocardial infarction, although considerable bronchospasm was noted. The following morning, the medical consultant felt that respiratory failure was present, this was confirmed within a few minutes with a pH of 7.0 and pCO_2 of 120 mm Hg. An endotracheal tube was passed, the patient was placed on a Bird respirator, Isuprel being added every four hours. See Table 2 for serial pH and pCO_2 values. Secretions, severe bronchospasm (requiring curarization), hypotension and later,

Table 2

Date	Peak Flow	pH	pCO_2	CO_2 Content	Comment
7/13	105L	7.32	48mmHg	26mEq	
7/15					IPPB & Isuprel
7/16					T.U.R.
7/18	185				
7/24		7.00	120	36	coma
		7.07	120		
		7.18	82	32	
7/25		7.27	72	34	responds simple commands
7/26					tracheotomy
7/27		7.37	58	34	
7/29		7.28	80	38	
8/2					febrille, coma
8/3		7.56	40	36	hypothermic
8/6					expired

inability of patient to ventilate adequately at any time on his own were problems dealt with until his demise 13 days following this acute problem. During his last 72 hours the temperature fell below 94°F. and was associated with hypotension and unresponsiveness, suggesting brainstem damage. He had been responding to simple commands up until 3 days before his demise. Two serum lactic dehydrogenase studies were elevated, suggesting pulmonary infarction, a chest film (portable) did not add confirmatory evidence. Serial ECG's showed no evidence of myocardial damage. Urine output remained over 2 liters/day. Serial pCO_2's and pH's were used to determine the adequacy of alveolar ventilation particularly when bronchospasm was severe. Also measuring the expired volume from the Bird respirator (tidal volume) provided a good index of ventilation and degree of bronchospasm.

The episode of acute respiratory acidosis and coma may have been the result of a pulmonary embolus, atelectasis or some other factor. Even if it had been a large pulmonary embolus or infarct, this in itself would not have resulted in such severe CO_2 retention and acidosis. Inadequate ventilation – due to severe bronchospasm, secretions and hypoxia (all of which may have been triggered by embolus, atelectasis, etc.) was the life threatening event which had to be recognized and reversed. A serum CO_2 would not have indicated the severity of the problem, the pCO_2 did. Adequacy of ventilation could not be ascertained by auscultation. The pCO_2 did not begin to drop following intubation, suctioning and artificial respiration until finally curarization was instituted, as he had failed to improve on Heparin, corticosteroids and Isuprel. Whether there was sufficient cerebral hypoxia with the initial acute episode to result in permanent brain damage is unknown, but was suggested by his behavior after blood gas studies had returned to normal.

This history illustrates how well surgery was tolerated initially with pre- and postoperative IPPB and Isuprel, in a patient with severe obstructive pulmonary disease and hypoventilation. The final major complication of acute respiratory failure was a late complication of surgery and inactivity, apparently due to a pulmonary embolus, superimposed on severely diseased lungs. No post-mortem was permitted.

ACKNOWLEDGMENT

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Program-In-Brief For M.M.A. Annual Session

Pages 117 and 118

Special Report

AMA Blood Bank Conference

FRANK W. KIBBE, M.D.

At the first Blood Bank Conference sponsored by the American Medical Association and held in Chicago in December 1964, several general points of interest were propagated. I will enlarge somewhat on each of these points but the following were the main areas of discussion:

1. Types of banks and their advantages
2. Donor recruitment
3. Government control at National and State levels
4. The Kansas City Controversy
5. Research aspects

1. Types of banks and their advantages:

Each speaker setting out his program seemed quite convinced of its merits. Roughly these programs divide into:

A. Regional banks, either M.D. and hospital sponsored or Red Cross controlled. These, in general, acted for moderately large areas, up to 200 miles in diameter, had a central processing laboratory and mobile units for collection and/or distribution of blood. They had the great advantage of good laboratory control, and less outdating as blood could be shifted from one hospital to another on demand. Dr. C. Y. Gates,[1] who presented the paper for such a bank under the guidance of the San Francisco Medical Society, showed excellent coverage and highly successful voluntary recruitment.

Dr. Charles C. Lund[2] spoke on the Red Cross system in Massachusetts and how successful it was in covering the state's needs. From his tables one would assume that the Red Cross fills in the needs rather than supplies them completely.

B. Community Banks: Each of these serves a number of hospitals in smaller circumferential areas, say within a city. Each has a central laboratory which is the donor point and both processes and stores blood. Mobile units are used only to transport the blood to and from hospitals for usage. Usually these are set up by a group of pathologists in local hospitals, but in some instances they may be of the Assurance type as exemplified by the Southwest Blood Banks,[3] Inc. of Scottsdale, Arizona.

Here a member may join the group by payment of a fee of either one or two dollars with the understanding that he will be called on to donate blood about once every three years. The group has actuarial figures showing that each 100 families in the group require about 11 pints a year and that this amount is sufficient also to cover older persons having no family and who are unable to donate themselves. Such persons can join at a slightly higher fee.

C. Local hospital banks. These function as much as possible on a voluntary basis and may for convenience utilize the AABB Clearing House method of collection and reimbursement. However, this does not allow for actual exchange of blood but only for crediting and debiting on paper. Unless a hospital is large and transfuses 1000 pints or more a year, the problem of outdating may become a major one.

D. Commercial Banks. Here blood is bought, processed and sold as required. Some of these are AABB approved and meet exacting requirements, others are not. These banks fulfill a definite need and are particularly active in large centers. In the smaller areas a few rare blood-type donors are listed as being professional donors. Many of these may be paid for a specific donation, but most are willing to give when asked with or without recompense.

2. Donor Recruitment

Local campaigns, diffusion of knowledge, hospital pressure and physician cooperation are the basic ingredients of a good voluntary donor program. By approaching groups, be they industrial or social, one can usually obtain good cooperation and if the pathologist and laboratory technicians offer free typing facilities to such organizations, it tends to swell the donor lists at the hospital and make available both larger quantities and rarer types of blood.

As has been stated[4] over and over again the problem of Homologous Serum Jaundice increases markedly with the Commercial Bank Blood and the non-voluntary donor. Reports vary but the number of cases is in the neighborhood of 5-10 times as great from such paid sources. While the SGPT determination is helpful in screening out active hepatitis there is no specific test for the presence of the hepatitis virus and until there is, the use of a good history with a voluntary donor is our best safeguard.

3. Government Control

Whereas physicians for the most part, have regarded the giving of whole blood to a patient as a "service," there have been increasing pressures by governmental agencies to regard such blood as a "commodity." Such concern has given rise to necessary legislation pertaining to blood derivatives such as: gamma globulin, albumin and fibrinogen. Here it is possible to set standards for control of sterility, purity and activity as is true with any medication. But, to date, in the case of whole blood, it is only by history from and tests on the donor that the quality of the blood can be established. In the field of whole blood the National Institutes of Health now license commercial blood banks and control shipment of blood over state lines.

Within the states themselves some laws have been enacted concerning blood banks and their activities. There remains a tendency among pathologists to consider their own policing of the laboratory as superior to that of someone without medical training, and this seems quite reasonable.

4. The Kansas City Decision

In Kansas City, the situation of quality control of a Commercial Bank run by a non-professional and his wife versus the quality control of a community bank run by hospital personnel with pathologist supervision does *NOT* enter into the legal controversy.[5] The Federal Trade Commission bases its case on a "restraint of trade" wherein the community bank and its members have allegedly conspired to refuse the commercial bank's output and thus attempted to put the latter out of business. The defense of the pathologists involved must lie with their SEPARATE beliefs that the refusal of blood from this NIH licensed bank had to do with patient care and the avoidance of an inferior unit of blood.

No matter how this controversy is settled, it is completely within the powers of the FTC to initiate proceedings, under existing anti-trust laws, against any hospital or bank refusing replacement by an NIH licensed bank despite a hospital's belief that it is receiving inferior goods. Again this stems from the word "product" or "commodity" as opposed to "service."

At present, legislation proposed by U. S. Senator E. V. Long (Missouri) would exempt "not-for-profit" blood banks from the anti-trust laws and thus from FTC control. As yet the bill has not come up for vote and may be reintroduced.

In Maine we are faced with the problem of the unavailability of blood to small hospitals for emergency service. Along with this comes the "outdating" dilemma as regards smaller banks which attempt to maintain some kind of blood supply within the hospital and not depend entirely on the so-called "walking bank."

5. Research aspects

Research such as Dr. Charles Huggins[6] has carried out with frozen blood may supply an answer to storage of blood for long periods and thus keep available supplies for shipment throughout the state. But the problem of frozen blood is that it requires the expensive Cytoagglomerator, not only to nurse it into the frozen state but also to guide its withdrawal from this state into readiness for transfusion. Perhaps a permanent Cytoagglomerator with a low temperature freeze-hold storage plant in a central area plus mobile Cytoaggomerators to move and process the blood at small hospitals would be possible but this is not a practical approach.

Two other answers to our problem seem more feasible. The two are not completely interdependent, but do complement one another: 1. The recent development of a new phosphate anti-coagulant (C.P.D.)[7] to use with the blood instead of the A.C.D. solution we now have allows longer storage of refrigerated blood. This, if once licensed by NIH would allow an extra week of storage of whole blood with equal or better red cell survival, thus reducing the outdating concern. Dr. Joseph Porter[8] of our own Maine Medical Center has done some experimental work with this solution and has reported it to be most satisfactory. 2. The increased movement of units of blood from one hospital to another, into and out of larger banks and centers via fitted mobile units could be an answer to most area shortages. This might obviate the necessity of purchasing blood from outside commercial sources either in or out of state because smaller hospitals could act as drawing areas and could ship the blood to the larger centers for processing and use. Some of the regional banks move blood as far as 200 miles and I am certain that movement in and around cities as Portland, Waterville and Bangor would not involve such distances. The age of blood that can be moved may be a controversial point; however, with the newer anti-coagulant mentioned above it is likely that older blood could be moved more safely without the danger of hemolysis. Correctly stabilized containers with refrigeration housed in the mobile busses might well make the well-traveled blood quite as safe as that stored within hospital vaults.

These centrally located laboratories are also equipped to remove plasma from outdated or unusable blood and send this to any of several commercial drug houses. In turn, they would receive credit in fresh plasma which could be held at these laboratories or shipped via the carriers to local hospitals for storage against emergency. This plasma can be held at room temperature and over a period of six months, loses the danger of transmitting the hepatitis virus. Such plasma could well be a temporary answer to some emergency crises while awaiting the delivery of whole blood. In conclusion, I hope that some positive action will be taken by the many hospitals of Maine to effect such exchanges to improve medical, and especially emergency care, as well as to minimize the necessity for commercial invasion of this well-motivated voluntary service.

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2. Charles C. Lund, M.D.: Massachusetts Regional Blood Program of American Red Cross.
3. W. Quinn Jordan: Blood Assurance Plans, Southwest Blood Banks, Inc.
4. Charles B. Wheeler, Jr.: State Laws and Regulation.
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6. Charles E. Huggins, M.D.: Blood Research Highlights.
7. Lewis Diamond, M.D.: History of Blood Banking in United States.
8. Joseph Porter, M.D.: Personal Communication.

R.F.D., Lincolnville, Maine

Maine Medical Association

Program-in-Brief – 112th Annual Session

The Samoset – Rockland, Maine

Sunday – Monday – Tuesday

June 13, 14, 15, 1965

Sunday, June 13

9:30 A.M. First Meeting of the House of Delegates

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

6:30 P.M. Dinner
Speaker: To be announced

Monday, June 14

9:45 A.M. **Some Clues in the Care of Cardiacs**
SAMUEL A. LEVINE, M.D., Consultant in Cardiology, Peter Bent Brigham Hospital, Brookline, Massachusetts

11:00 A.M. **The Role of Simple Mastectomy in the Treatment of Cancer of the Breast**
GEORGE CRILE, JR., M.D., Head of the Department of General Surgery, Cleveland Clinic Foundation, Cleveland, Ohio

12:00 NOON to 2:00 P.M. Luncheon

2:00 to 4:00 P.M.
Scientific Program Sponsored by the Maine Chapter, American College of Surgeons and Maine Trauma Committee

2:00 P.M. Subject to be announced
GEORGE CRILE, JR., M.D., Cleveland, Ohio

3:00 P.M. **Panel – Problems of a Case of Multiple Trauma**
Moderator: ROBERT H. KENNEDY, M.D., Director of Field Programs, Committee on Trauma, American College of Surgeons, New York
Panel:
CHARLES A. HANNIGAN, M.D., Internist, Auburn
H. RICHARD HORNBERGER, M.D., Surgeon, Waterville
JOHN R. LINCOLN, M.D., Anesthesiologist, Portland
JOHN A. WOODCOCK, M.D., Orthopedist, Bangor

4:00 P.M. Election of President-elect

6:00 to 7:00 P.M. Social Hour, Dutch Treat

7:00 P.M. Annual Banquet
Speaker: J. M. HAYMAN, JR., M.D., Dean, Tufts University School of Medicine, Boston, Massachusetts
Subject: **Trends in Medical Education**
Presentation of Honorary Pins

Tuesday, June 15

9:30 A.M. to 12:00 NOON

9:30 A.M. **The Medical Treatment of Rheumatoid Arthritis**
KENNETH FREMONT-SMITH, M.D., Junior Associate in Medicine, Peter Bent Brigham Hospital, Brookline, Massachusetts

10:30 A.M. Panel - **Your Children and Mine, Their Problems From 12 to 20**
Moderator: J. ROSWELL GALLAGHER, M.D., Chief, The Adolescents' Unit, The Children's Hospital Medical Center, Boston, Massachusetts
Panel:
ACHSA BEAN, M.D., Owl's Head
WILLIAM E. SCHUMACHER, M.D., Augusta

12:00 NOON to 2:00 P.M. Luncheon

2:00 to 4:00 P.M.
Program Sponsored by the Maine Medico-Legal Society

2:00 P.M. **Psychiatry and Criminal Responsibility**
PETER W. BOWMAN, M.D., Superintendent, Pineland Hospital and Training Center, Pownal

3:00 P.M. **The Attorney General's Office and the Investigation of Crimes in Maine**
The Honorable RICHARD J. DUBORD, Attorney General of the State of Maine

6:30 P.M. Clam Bake

SPECIALTY GROUP MEETINGS

Monday, June 14

2:00 to 4:00 P.M.

Maine Thoracic Society and Maine Society of Internal Medicine

Medical Trends Today - Your Masters or Your Servants?

FRANK P. FOSTER, M.D., Member, Board of Trustees, American Society of Internal Medicine, Boston, Massachusetts

Guide Lines For Treatment of Acute Respiratory Insufficiencies in Chronic Pulmonary Patients

MAURICIO J. DULFANO, M.D., Assistant Professor of Medicine, Tufts University School of Medicine, Boston, Massachusetts

Maine Society of Clinical Hypnosis

Hypnotic Technics in Therapy

CALVERT STEIN, M.D., American Board of Psychiatry and Neurology, Springfield, Massachusetts

Maine Society of Obstetrics and Gynecology

Maine Society of Anesthesiology

Tuesday, June 15

10:00 A.M. Maine Medico-Legal Society

Business Meeting

Presiding, IRVING I. GOODOF, M.D., Waterville

2:00 to 4:00 P.M.

Ear, Nose and Throat Group

Recent Advances in the Surgical Approach to Sinus Disease

WILLIAM W. MONTGOMERY, M.D., Associate Professor, Harvard Medical School, Boston, Massachusetts

Maine Radiological Society

Section on Ophthalmology

Maine Chapter of the American Academy of Pediatrics

Annual Meeting

Presiding, RUSSELL A. MORISSETTE, M.D., Lewiston

SPECIAL NOTICES

Sponsors

The speakers for the scientific programs are supported in part by grants from Merck Sharp & Dohme Postgraduate Program, Eli Lilly and Company State Medical Convention Program, Geigy Pharmaceuticals, the Maine Cancer Society and The Maine Heart Association, Inc.

Election of President-elect

The Election of a President-elect will take place at the General Assembly, June 14 at 4:00 p.m.

House of Delegates

Sunday, June 13 - 9:30 a.m. and 3:00 p.m.
Included in the Order of Business for the meetings of the House of Delegates will be final action on the Budget for 1966, Standing and Special Committee Reports, and the Nominating Committee Report.

Election of Councilors

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 13 at 3:00 p.m.

Third District — Knox and Lincoln-Sagadahoc

Fourth District — Kennebec, Somerset and Waldo

Golf Tournament

Daniel R. Shields, M.D., Chairman

The Journal of the Maine Medical Association

DANIEL F. HANLEY, M.D., Brunswick, Editor

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Maine Hospital Association

FREDERICK T. HILL, M.D., Waterville — PEARL R. FISHER, R.N., Waterville

Across The Desk

The Nurses Are Coming, Hurrah Hurrah!

Admissions to schools of nursing in the United States rose to an all time high of 92,300 students in 1964, Fred C. Foy, chairman, Committee on Careers, National League for Nursing, New York, announced recently. That figure represents an increase of 10,400, or 12.7 percent, over 1963. The rise can be attributed in large part to the post World War II "baby boom," which is expected to bring 63 percent more college-age students into higher education by 1975, Mr. Foy explained.

The most marked change in nursing was in admissions to the 1,158 programs of professional nursing. These reached an estimated 56,413 for the calendar year 1964, a breakthrough from the plateau-level of 51,000 which has prevailed over the past few years. Of these, the largest number of new students, 38,986, were admitted to three-year diploma schools in hospitals. Second came 11,515 young men and women accepted by four-year baccalaureate programs in colleges and universities. The remaining 5,912 entered two-year junior or community college programs in nursing, which lead to an associate degree.

Coronary Care Units

"Coronary Care Units," the first publication to describe specialized intensive care units for acute myocardial infarction patients, has been issued by the Heart Disease Control Program of the Public Health Service, U. S. Department of Health, Education, and Welfare.

The new 40-page brochure is directed to hospital administrators and health professionals concerned with hospital care following heart attacks. It covers new concepts of 'round-the-clock electronic monitoring, staffing patterns for medical and nursing personnel, and outlines the nurse training required for effective service in the specialized unit. As a further aid in planning, it includes suggested designs for 4- or 5-bed coronary care units and discusses existing equipment.

The brochure indicates that cardiac standstill and potentially lethal disturbances of heart rhythm can be detected and treated promptly in coronary care units. It is believed that the salvaging of "hearts too good to die" can substantially reduce the present post-infarction death rate, which currently runs to nearly one-third of hospitalized cases.

Single copies of "Coronary Care Units" are available without charge from the Public Health Service, Washington, D. C. 20201. Copies may also be purchased from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 20402, at twenty cents a copy.

Giving The Rats "The Business"

The rats developed ulcers when they were given massive doses (about 800 times the human dose) of reserpine, a drug normally used for treating high blood pressure, and then subjected to stress. This parallels nearly the way that ulcers are developed in some humans — by stress. Because rats have no business worries to produce stress, mild electrical shocks were used to do this.

Conscious Without Pain

A University of Michigan medical research team said recently a new compound, CI-581, can produce "profound analgesia and short-lasting anesthesia."

Drs. Edward F. Domino, Peter Chodoff and Guenter Corssen, of the departments of pharmacology and anesthesiology, U. of M. Medical Center, reported on the first human pharmacologic studies of the new anesthetic candidate before the Federation of American Societies for Experimental Biology.

"These first studies of CI-581 indicate that intravenous

administration of the drug can produce profound analgesia and short-lasting anesthesia," Dr. Domino said. The powerful analgesic action is of special interest.

The University physicians reported that one of the most dramatic findings of the study was the "marked degree of analgesia without loss of consciousness which could be induced by CI-581.

"This was demonstrated dramatically when the drug was given as a continuous intravenous infusion . . . At approximately 125 to 180 drops per minute, the subject showed profound analgesia without loss of consciousness during the infusion period . . . and felt absolutely no pain."

Drug Reactions

The U. S. medical profession, the pharmaceutical industry and the Food and Drug Administration of the U. S. Department of Health, Education, and Welfare are cooperating to establish a major addition to existing adverse drug reaction reporting programs.

A Registry of Tissue Reactions to Drugs will be established within the Armed Forces Institute of Pathology (AFIP) in Washington. Joint sponsors are the American Medical Association, FDA and the Pharmaceutical Manufacturers Association. The AFIP has the world's largest repository of pathological material for research and education.

"This unique cooperative effort for the benefit of the American public is the first between major drug manufacturing, drug prescribing and drug regulating bodies."

Facts About Hospitals

The average patient's hospital stay is nearly half as long today as it was 25 years ago. The average stay in 1935 was 14 days and today it is 7.7. The decline is attributed to better medicines, new equipment, and improved treatment.

Three-fourths of all drugs, antibiotics and vaccines in hospital use today were unknown ten years ago.

On the average, one in seven persons probably will be hospitalized sometime this year.

Nearly 1½ million persons are patients in hospitals on any given day. More than half of them (720,674) are in psychiatric hospitals.

The number of admissions of patients with psychiatric diagnoses to general hospitals is estimated to be as great as the number of patients admitted to state and county mental hospitals (342,483 to state and county psychiatric hospitals).

Hospitals rank as one of the nation's largest employers. Total employment in the 7,138 hospitals registered by the American Hospital Association in 1963 reached 1,840,287.

A general hospital employs 241 persons to care for every 100 patients. In 1946, the ratio was 148 employees for every 100 patients.

Total payroll expense of hospitals in 1963 reached $7.3 billion, accounting for 66.3 per cent of the total $11 billion expended by all hospitals.

Some large hospitals have 200 or more different job classifications.

Assets of U.S. hospitals in 1963 amounted to $21 billion, more than $17 billion of which was in physical plants.

More than 145 million persons – 77 per cent of the population – had some form of voluntary health insurance in 1963, nearly 3.9 million more than in the previous year.

Health insurance benefits paid insured persons for hospital-surgical-medical care in 1963 totaled $6.9 billion, an increase of $622 million over the previous year.

The number of births in hospitals declined from 3,857,626 in 1962 to 3,784,666 in 1963, reflecting a nationwide lowering in the birth rate.

Hospitals now deliver 97 out of every 100 births in the United States, compared to 37 of every 100 in 1935.

The average cost to the short-term general hospital for each day a patient spends in the hospital is $38.91. For that sum, the hospital provides the complete treatment, bed, food, and nursing service around-the-clock.

HAVE YOU MADE YOUR RESERVATION FOR THE ANNUAL SESSION

June 13, 14, 15, 1965

State Of Maine

Department of Health and Welfare

DEAN H. FISHER, M.D.
COMMISSIONER

Reporting of Abused or Battered Children

EDGAR J. MERRILL, M.S. in S.S.*

This article has been stimulated by a bill, L.D. 574, "An Act to Provide Protection for Physically Abused Children," which was before the 102nd Legislature. This was signed into Law by Governor Reed on March 11, 1965, becoming P.L. 68. The basic purpose of this law is to require mandatory reporting by physicians and institutions of those situations where there is reasonable *cause to believe* that a child under sixteen years of age has had physical injury or injuries inflicted upon him by other than accidental means.

This type of legislation is not new, though there are significant differences in the content of the many existing state laws. P.L. 68 appears to have most of the positive features of these many laws. Let us discuss some of these more positive features of this law.

Perhaps the first feature of the law which comes to attention is that both physicians and institutions are identified as having to report situations. This is significant because in Maine there are considerable differences from area to area as to the medical resources. In some areas there are large, well staffed hospitals with emergency clinics where many such children might be seen. P.L. 68 makes possible reporting by such hospitals, where the child was treated as an emergency, by representatives of the hospital. However, in many areas of the State without hospital facilities, or facilities with no staff physicians, individual doctors treating the child as a private patient would be reporting such instances. Under some state laws situations were not reported when the child appeared at the hospital because there was confusion as to whom should take the responsibility of reporting. This is clarified by P.L. 68.

Another important feature of P.L. 68 is that the agents to whom such reports shall be made are clearly spelled out. They are the Division of Child Welfare, Department of Health and Welfare, and the county attorney. Again, this eliminates any question or confusion as to the appropriate agency to become involved. The selection of these two agents appears to be a sound choice for several reasons. The Division of Child Welfare is administratively structured and currently providing services which lend themselves well to working in the area of physically abused children. District Offices of the Division are established across the State in such a way as to offer basic child welfare services to every community in the state. These district offices are administered by local directors (District Supervisors), and manned by a competent and capable staff of child welfare social workers. Normally, representatives of district offices have established working relations with local hospitals and many physicians. In addition to this, the staff of the Division of Child Welfare is currently offering intensive services to children in need of care and protection. Included among these children are a number who have been physically abused by an adult. Such referrals from hospitals or physicians, then, will not create a new program for the division, but will hopefully permit an on-going service to reach out to more children who appropriately require child protective service.

It was equally sound to identify a county attorney as the other agent to whom such referrals should be made. In many instances (though not all) physical abuse or child battering situations have to be brought before an appropriate court. Under existing circumstances the Division of Child Welfare and county attorneys work closely on matters concerning child protection. Relations with attorneys and courts are established, again, meaning that no new procedures or programs have to be developed to adequately serve children being referred under a law such as P.L. 68.

One final comment about this feature of to whom referral would be made. Under this statute the assigning of responsibility for child protection is specifically placed with three professions, all deeply rooted in service to people – social work, law and medicine. In any area of human life it is difficult, if not impossible, to identify any one profession or group of people who should be held totally responsible. Our society is so complex, and the needs of people and their behavior so varied that basic responsibility, of necessity, has to be assumed by many. This is especially true in this day of increasing specialization, both between and within the professions. Perhaps a brief discussion of the possible roles of law,

*Director, Division of Child Welfare

medicine and social work in the area of physical abuse would help establish this.

Under normal circumstances when a child is severely abused, medical care is required and medical personnel thus become one, if not the first, of the professions to become aware of a particular situation. After a decision is reached that there is cause to believe a child's injury was inflicted upon him, the situation is referred to a social work agency, the Division of Child Welfare. There follows a comprehensive social study and evaluation of the family, including causes for the behavior, prior history, probability of recurrence, and possible courses of action. If, on the basis of the social study, it is felt that protection of the child requires possible removal from the home, and action against an adult, the county attorney is consulted. If it appears that indeed legal action is needed, proper procedures are initiated, and the matter comes before the court. It is during this phase of the service to children, that the three professions again combine to protect the child. If the judicial decision is to remove the child from its home, in all probability the Department of Health and Welfare will be required to assume full responsibility for the child, thus ensuring continued care and protection. A brief case summary here will serve to illustrate the effectiveness of the three professions working together to assure the basic protection of one child.

A district office of the Division of Child Welfare was notified by the local hospital that an infant had been brought in that morning with severe bruises and lacerations over its face, head and abdomen with also a possibility of several broken ribs. The only reason the mother gave for this physical damage, was that the child had fallen. She insisted on taking the child home, though the hospital was convinced of the need for a period of hospitalization for treatment and further study. The child welfare worker telephoned the county attorney and together they spoke with the judge who issued a temporary commitment order. The Division of Child Welfare kept the child at the hospital.

During the next two weeks a social study was made including interviews with both parents. They maintained that all damage was accidental. During this period two additional facts came to light. One that the child had been to the hospital earlier in a similar condition, and that an x-ray showed healed skull fracture and broken ribs. The other fact was that all events leading to the physical damage occurred during the day when the mother was alone with the child. With this knowledge, another meeting was held with the county attorney who felt the evidence indicated the need for a formal hearing.

At the hearing parents continued to state only that the damage was accidental but being unable to give significant details. Results of the social study, and the medical findings, were presented. Also presented was the medical opinion that such physical damage was too excessive and extensive to be accounted for by a fall.

Based on this information, the child was committed to the Department of Health and Welfare. Though there was no admission of abuse, the fact that such severe and repeated damage did occur while in the care and custody of a parent was an indication of serious child neglect.

Another important feature of P.L. 68 is the section entitled immunity from liability. This section provides immunity from civil or criminal liability for persons either making an initial report under this statute, or who participate in a subsequent judicial proceeding. Perhaps to individual physicians or institutions this feature is one of the more important ones. This section should free medical personnel to make referrals on the basis of probable cause, without the necessity of having to prove beyond doubt that a specific individual inflicted specific injuries on a specific child. It is not the intent nor purpose of any mandatory reporting law to pose the determination of guilt or innocence onto either the medical person who makes initial referrals of such situations, nor onto the child welfare social worker whose function it is to evaluate and perhaps to counsel within the total family situation. Rather, the purpose is to initiate the service which will result in immediate protection for a given child and provide a future of continued protection, if needed. In fact, it is not necessarily the responsibility of the social agency through its evaluation, study and counselling efforts, to determine guilt or innocence. This is the function of the court.

In conclusion, there are several generalized comments which can be made. P.L. 68, and other state laws regarding child protection, are primarily aimed at protecting the rights of children. It is not primarily to bring to justice and initiate punishment of adults who violate children's rights. There are many other laws aimed at punishing adult offenders, and police departments and courts of law to enforce these laws, and make decisions of guilt or innocence. If the attitude and approach to laws protecting children's rights can remain centered on helping children – not punishing adults, on providing continuing protective environments for children – not establishing guilt or innocence of adults, there appears to be no reason why the professions of medicine, law and social work cannot function harmoniously. Certainly there are appropriate roles for these professions in helping children, and these roles are or can be clearly defined.

Maine Heart Association Notes

Changes In Cardiopulmonary Functions Related To Abstinence From Smoking

"Physiologic pulmonary changes associated with smoking are important from many points of view. - - - Many - - - studies have been concerned with the acute changes after the smoking of one or two cigarettes. We recently made measurements of lung volumes, ventilatory function, and some cardiopulmonary responses to exercise in a group of nonsmokers and found statistically significant differences between the two groups. - - - The present study was made in order to further evaluate the pulmonary alterations in young smokers and to determine their reversibility after short periods of abstention from smoking.

"- - - pulmonary function and some cardiopulmonary responses to exercise were measured before smoking was discontinued and after 3 and 6 weeks of abstinence from smoking in ten young chronic cigarette smokers.

"After 3 weeks of no smoking, expiratory peak flow and pulmonary diffusing capacity were significantly increased, and - - - oxygen debt - - - was significant decreased. After 6 weeks of no smoking, - - - inspiratory reserve volume and maximal voluntary ventilation were increased, and airway resistance was significantly decreased - - -.

"This gives further evidence that alterations in lung function are present in young smokers and that some of the alterations are quickly reversible in the young cigarette smoker with abstinence from smoking."

Submitted by Jacob B. Dana, M.D.

Reference: Krumholz, R. A. et al. Annals of Internal Medicine, Volume 62, pages 197-207, 1965.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Liver Perfusion

BOSTON, MASS. — Lahey Clinic Foundation scientists have checked up on the first two years of their experiments with a pocket-size pump which continuously infuses drugs into cancerous livers.

They found that two out of three patients healthy enough to take the treatment were helped.

The results of this research were disclosed by the American Cancer Society which helped support the work. The scientists are Dr. Robert D. Sullivan, who conceived of the treatment, Dr. Elton Watkins, Jr., who developed the pump, and Drs. Wladyslaw Z. Zurek and Amir M. Khazei.

Patients whose cancers originate in or spread to the liver are considered beyond cure. A few gifted surgeons have removed cancerous liver lobes, but it is doubtful whether this heroic technique helped many patients for an appreciable period.

This grim fact led the Lahey group more than two years ago to institute a new procedure in which drugs would be dripped into the cancerous livers continuously for long periods. Inasmuch as cancer cells are vulnerable to a drug only when they are dividing, continuous treatment conceivably might knock out all the cancer cells.

The new procedure goes like this:

1) At operation, surgeons cut out whatever cancer is removable;

2) they then insert the nozzle of a long stringy Teflon catheter into the hepatic artery which brings blood to the liver and sew it firmly into the artery wall;

3) a one-and-one-half foot (1½′) length of the catheter is left outside the body when the belly wall is closed;

4) a pump is attached to the end of the catheter, and it pumps anti-cancer drugs through the catheter into the hepatic artery where the blood stream carries it in countless branches through the diseased liver; because the liver sops it up and detoxifies it, very little of the strong drug gets back into the general circulation.

The 12-ounce pump is about the size of a hearing aid and fits into a coat pocket. Operating on a clock mechanism, it is wound every eight hours. A disposable plastic container holds 25 cc (five-sixths of a fluid ounce) of the drug which is pumped drop by drop, day and night for five days into the system. When the five-day supply is exhausted, the patient discards the container and inserts a full one into the pump.

Ambulant patients receive constant medication while they work, play and sleep. The infusion can be stopped and started at will, and the drug can be changed merely by putting a container of another medication in the pump.

During the first two years, a total of 78 patients with liver cancer were treated by the pump. Of these, 60 were in good enough shape to live until the medication could have effect. Liver cancers shrank and sustained benefit was observed in 41 patients.

Patients' livers were infused continuously for anywhere from nine to 240 days. The average infusion lasted 40 days. While several drugs have been tested, the most satisfactory appear to be 5-FU (5-fluorouracil) and 5-FUDR (5-fluoro-2′-deoxyuridine).

In some patients, liver tissues became inflamed after long periods of infusion. In these cases, distilled water was substituted for the drug, and the inflammation subsided after two or three weeks.

The Lahey group feel that while continuous infusion may never cure liver cancer, it affords substantial benefit to a majority of the patients who survive long enough to be treated. In 21 patients, cancer signs disappeared completely for from two to 17 months to date after treatment had ended, and 15 at this time still are without evident regrowth of their disease. No patient is considered cured, however.

From American Cancer Society News Service Release

Recommended Dosage of Lomotil Liquid to Control Diarrhea

File: Diarrhea

LOMOTIL®

Age	INITIAL LOMOTIL LIQUID DOSAGE*
3-6 mo.	½ tsp. t.i.d. (3 mg.)
6-12 mo.	½ tsp. q.i.d. (4 mg.)
1-2 yr.	½ tsp. 5 times daily (5 mg.)
2-5 yr.	1 tsp. t.i.d. (6 mg.)
5-8 yr.	1 tsp. q.i.d. (8 mg.)
8-12 yr.	1 tsp. 5 times daily (10 mg.)
Adult	2 tsp. 5 times daily (20 mg.) (or 2 tablets q.i.d.)

*Based on 4 cc. per average teaspoonful.
Note: After diarrhea is controlled the initial dosage can usually be reduced to meet the requirements of the individual patient.

LOMOTIL® TABLETS/LIQUID

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride .....2.5 mg.
(Warning: May be habit forming)
atropine sulfate .....0.025 mg.

Precautions
Lomotil is an exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Cautions and Side Effects
Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdosage.

SEARLE *Research in the Service of Medicine*

County Society Notes

ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at the Central Maine General Hospital in Lewiston, Maine on February 18, 1965.

The meeting was called to order by the President, Henry C. Thacher, M.D.

A motion presented by Merrill S. F. Greene, M.D. that the county society hold a minimum of six meetings a year was tabled until the March meeting.

A meeting of the Androscoggin County Medical Association was held at St. Mary's General Hospital in Lewiston, Maine on March 18, 1965 with twenty members present.

The meeting was called to order by the President, Henry C. Thacher, M.D. It was voted to change Article IV — Sessions and Meetings of the bylaws to read Section 1. "The Association to have a minimum of six (6) meetings per year."

The guest speaker Walter Rohm, M.D. of Augusta, Director of female service and extramural services of the State Mental Health Department, was introduced by Harvey J. Proulx, M.D. Dr. Rohm's topic was Psychiatric Care in a Community Hospital. He outlined the program as it was set up in Manitoba. This was followed by a round table discussion of some of the inherent problems in setting up a Mental Health and Psychiatric Unit in conjunction with a community or general hospital. Reuben Leitman, M.D. discussed some of the problems and shortcomings of admission to a general hospital. There were advantages which were presented.

DONALD L. ANDERSON, M.D.
Secretary

KENNEBEC

The Kennebec County Medical Association met at The Jefferson in Waterville, Maine on March 18, 1965. The meeting was called to order by the President, John D. Denison, M.D.

Paul H. Pfeiffer, M.D. discussed the current status of the "Eldercare" and "Medicare" controversy.

Edward C. Matthews, M.D., practicing pediatric cardiology in Portland, was the guest speaker. Dr. Matthews gave a clear discussion of congenital cardiac defects, illustrating their causes, diagnosis and management by using x-rays and phonocardiograms along with routine electrocardiograms.

EARLE M. DAVIS, M.D.
Secretary

CUMBERLAND

The Cumberland County Medical Society met at Valle's Steak House in Portland, Maine on March 18, 1965 with eighty-five members and guests attending.

Following a social hour and dinner, the President, Alvin A. Morrison, M.D. announced that the resolutions and suggestions of the society in regard to a unified medical complex had been relayed to the subcommittee of the Medical Advisory Committee for presentation to the hospitals involved. Dr. Morrison acknowledged receipt of an AMA publication pertaining to health facility planning which might serve as a guide to future medical society action. The Secretary, Stanley B. Sylvester, M.D., summarized Dr. Ronald A. Bettle's letter concerning public acceptance of a Brunswick radio program devoted to health information.

It was voted that the Health Careers Committee use its discretion in formulating a suitable program for the pre-medical clubs with utilization of the membership of the society as needed.

STANLEY B. SYLVESTER, M.D.
Secretary

LINCOLN-SAGADAHOC

The Lincoln-Sagadahoc County Medical Society met at The Ledges in Wiscasset, Maine on March 16, 1965 with twenty-four members and guests present.

John F. Dougherty, M.D. presented a synopsis of the last meeting of the Council of the Maine Medical Association.

A film *The Doctor Defendent* was shown and Mr. Robert W. O'Connor, counsel to the Maine Medical Association, led a discussion of prevention and defense of malpractice action in law. The five members of the Bar Association present joined the general discussion.

Mr. O'Connor also urged that members apprise their legislators of their views on bills coming before the state legislature.

GEORGE W. BOSTWICK, M.D.
Secretary

YORK

A meeting of the York County Medical Society was held at the Webber Hospital in Biddeford, Maine on March 10, 1965 with forty-two members and guests present.

Following a social hour and dinner, the meeting was called to order by the President, Alvin A. Hoffman, M.D.

The guest speaker of the evening, Richard H. Overholt, M.D. of Boston, Massachusetts, gave a very interesting talk on Glomectomy for Asthma which was illustrated by slides.

Thomas A. Martin, M.D., President of the Maine Medical Association, and Paul S. Hill, Jr., M.D., Councilor for the First District, spoke briefly concerning state and county society matters.

CHARLES W. KINGHORN, M.D.
Secretary

HAVE YOU SET ASIDE THESE DATES . . .

June 13, 14, 15, 1965—M.M.A. Annual Session

Necrologies

Theodore C. Bramhall, M.D.
1900-1964

Theodore C. Bramhall, M.D., 64, of Sarasota, Florida died on September 3, 1964. He was born in Belfast, Maine on June 11, 1900, son of Eben F. and Alice A. Bramhall.

Dr. Bramhall graduated from Colby College in 1924 and received his medical degree from Harvard Medical School in 1928. He interned as a surgeon at the Boston City Hospital for one year and was resident surgeon there for another year. He also served a residency in obstetrics at the Boston Lying-In Hospital and in gynecology at the Free Hospital for Women from 1930 to 1931.

Dr. Bramhall moved to Portland in 1931 and that same year joined the attending staff at the Maine General Hospital. He was chief of the gynecology service at the Maine General Hospital from 1945 to 1955 and in 1959 was elevated to the Honor Staff of the Maine Medical Center. He retired from active practice and moved to Sarasota, Florida in 1959 where he remained until his death.

He was an affiliate member of the Cumberland County Medical Society and the Maine Medical Association, a member of the American Medical Association and the Portland Medical Club. He was a Fellow of the American College of Surgeons, a member of the New England Surgical Society, the New England Obstetrical and Gynecological Society and the Boston Obstetrical Society. He was also on the consulting staff of hospitals in Bath, Belfast, Biddeford and Boothbay Harbor.

Dr. Bramhall is survived by his widow, Mrs. Elinor Strickland Bramhall; a son, Peter T. C. Bramhall of Falmouth, Maine and a daughter, Mrs. Priscilla B. Hodges of West Hartford, Connecticut.

Merrill J. King, Sr., M.D.
1894-1965

Merrill J. King, Sr., M.D., 71, of Portland, Maine died on March 18, 1965. He was born in Whitewater, Wisconsin on March 18, 1894, son of Harry H. and Hettie King.

Dr. King graduated from Lake Forest College in 1915 and received his medical degree from the University of Pennsylvania in 1919. He interned at the Misericordia Hospital in Philadelphia from 1919 to 1920 and served a residency at the Massachusetts Eye and Ear Infirmary from 1930 to 1932.

Dr. King practiced medicine at the Massachusetts Eye and Ear Infirmary from 1932 to 1954, moved to Rockland in 1954 to enter semiretirement and then came to Portland in 1963, where he served as a consultant until his death.

He was a senior member of the Maine Medical Association and the Knox County Medical Society and a member of the American Medical Association. He served as secretary-treasurer of the American Board of Ophthalmology and was a consulting surgeon of the Massachusetts Eye and Ear Infirmary.

Surviving are his widow, Mrs. Dorothy Bell King; a son, Dr. Merrill J. King, Jr. of Rockland; a daughter, Mrs. Parker Heath, Jr. of Concord, Massachusetts and seven grandchildren.

Herbert C. Scribner, M.D.
1889-1965

Herbert C. Scribner, M.D., 76, of Bangor, Maine died on March 18, 1965. He was born in Brewer, Maine on February 9, 1889, son of Mr. and Mrs. Herbert L. Scribner.

Dr. Scribner graduated from Bangor High School in 1907, from Holy Cross College and received his medical degree from Bowdoin Medical School in 1914. He interned at the Eastern Maine General Hospital in Bangor from 1914 to 1916.

Dr. Scribner practiced medicine and surgery at the Eastern Maine General Hospital and the Bangor State Hospital from 1916 until his death and served as Penobscot County medical examiner from 1922 to 1952.

He was secretary of the Penobscot County Medical Association for eighteen years, was an Honorary member of that Association and of the Maine Medical Association, having received a 50-year pin in June 1964. He was also a member of the American Medical Association.

Surviving are his widow, Mrs. Ethel Scribner; two sons, Charles Scribner of Hartford, Connecticut and Dr. Robert Scribner of Melrose, Massachusetts.

Charles E. G. Shannon, M.D.
1875-1965

Charles E. G. Shannon, M.D., 89, of Waterville, Maine died on February 2, 1965. He was born in Saco, Maine on September 16, 1875, son of Charles Way and Mary L. Shannon.

Dr. Shannon graduated from Thornton Academy in 1895, Colby College in 1899 and received his medical degree from Jefferson Medical College in 1902. He interned at Pottsville Hospital from 1902 to 1903 and served a residency at the Boston Eye and Ear Infirmary from 1903 to 1904.

He practiced medicine at the Philadelphia General Hospital from 1904 to 1919, Jefferson Medical Hospital from 1904 to 1949 where he served as Professor of Ophthalmology from 1927 to 1949. He moved to Waterville in 1949 and practiced there until his retirement from active practice in 1963.

Dr. Shannon was an Honorary member of the Kennebec County Medical Association and the Maine Medical Association, having received a 50-year pin in 1953, a 55-year pin in 1958 and a 60-year pin in 1963. He was also a member of the American Medical Association.

Albert C. Todd, M.D.
1911-1964

Albert C. Todd, M.D., 53, of Brewer, Maine died on December 7, 1964. He was born in Caribou, Maine on March 5, 1911, son of Herbert L. and Cora W. Todd.

Dr. Todd graduated from Caribou High School, Boston University in 1931 and received his medical degree from Boston University School of Medicine in 1934. He interned at the Eastern Maine General Hospital from 1934 to 1935.

Dr. Todd was a past president of the Penobscot County Medical Association, a member of the Maine Medical Association and the American Medical Association. He was chairman of the general surgery section and a member of the executive committee of the St. Joseph Hospital medical staff. He was also active in the Masonic order and a 32nd Degree Mason, a member of St. Andrew's Lodge, AF and AM of Bangor, Anah Temple, Order of the Mystic Shrine and the Colonel Brewer Lodge, Knights of Pythias of Brewer, a charter member of the Brewer Kiwanis Club and a trustee of the Brewer Savings Bank.

Surviving are his widow, Mrs. Sylvia P. Todd; a daughter, Mrs. Norma Dodge of Topeka, Kansas; a son, Paul W. of Berkeley, California; a sister, Mrs. Thelma Russell of Caribou; three grandchildren and several nieces and nephews.

Letters to the Editor

The Pros and Cons of General Practice

Perhaps this should come under the heading "Speaking Out," but the article "Who's Killing General Practice – Is General Practice Necessary" which appeared in the February 1965 issue of *The Journal of the Maine Medical Association* by Paul H. Pfeiffer, M.D. needs elaboration. It seems inconceivable that anyone seriously questions the need for good general practitioners. Certainly, the general public will agree with Dr. Pfeiffer in this, as does much of the medical profession. However, the definition of a good general practitioner as elaborated by Dr. Pfeiffer is unfair to half the physicians in this state, very few of whom have had any general practice residency. Medical training should equip all physicians to treat 90% of the illnesses and 60% of the ailments to which people are subject. This training can be obtained by four years of medical school and one year in a good rotating internship. Less general training than this produces limited physicians like the internist who cannot do a cutdown or tracheostomy when necessary or the surgeon who cannot differentiate a frank MI from simple left bundle branch block on electrocardiogram. Training beyond this should enlarge one's scope of treatment, but usually narrows it, because of limiting the types of patients seen. General practice residencies will always go begging for applicants because they are primarily designed to enable the applicant to reach the point in training where he should be before he applies.

The qualifications for a general practitioner should include the necessary training to make them competent, which in some instances would include years of additional training beyond internship, but usually not. More important are intellectual integrity, dedication and responsibility, which cannot be gained by additional training. A physician who performs an adequate history and physical examination, uses laboratory tests judiciously, keeps adequate records, obtains consultations when questions arise, makes himself available to his patients as much of the time as possible, secures adequate coverage with immediate availability of his records when he must be gone, maintains a definite program of continued education, and is pleasant and understanding with his patients, will provide the service which most patients desire and give them very sound medical care.

There are a great number of general practitioners of this type around. The services which they provide can never be replaced satisfactorily by clinics, groups or specialists.

Statements of additional years of training needed to produce a "good general practitioner" does the profession harm. It makes medical students feel that residency is a necessary part of medical training rather than a process of specialization. It also allows medical schools to go further from basic medicine with the feeling that the actual practice of medicine is learned in residencies. Several studies and pilot programs are underway to shorten the time spent in medical education. The six year combination college-medical school programs will have their primary purpose defeated if the two years are automatically added to the end of their training.

Most medical schools now plan to have their graduates thoroughly versed in basic principles and have them reasonably competent in working up patients. Some additional instruction in working *with* patients is still needed. Most rotating internships train the physicians to be competent in diagnosis in most fields, with awareness of the cases which require consultation. They also give enough surgical experience so that the graduating intern is a creditable surgical assistant and able to perform minor surgery and normal obstetrics – competently. Less training and one should not pass for a physician. More training is needed only for those who lack confidence in their ability to do as well as they have been taught, or those who wish to specialize.

A. DEWEY RICHARDS, M.D.
Bridgton, Maine

In reply to Dr. Richards letter I would like to state that he raises an important issue.

The article I submitted to the Journal discussed the pros and cons of general practice in response to an article by a Dr. Converse (pseudonym) in *Medical Economics*. Both Dr. Richards and I agree that generalists are a necessary part of the medical community.

The issue which Dr. Richards raises is whether or not training for the general man should take one or two years. He favors the traditional one year rotating internship which has trained many physicians who have contributed excellent service to their community. One problem with the "rotating" is its emphasis on surgery in the face of (1) a plethora of general surgeons who usually get another surgeon to assist them and, (2) the trend to disallow surgical privileges to all but those who have endured a surgical residency.

I would favor a rotating internship with emphasis on medicine, pediatrics and psychiatry with minor stress on obstetrics, minor surgery and medical EENT. One year is probably not long enough to make a man competent in these fields or to give him the confidence to set up shop in a small town where he would be largely on his own.

As for medical training being too long, I agree. However, it would seem more logical to curtail premedical training and basic science teaching and add on to the clinical teachings on the ward as a house officer. This is where medicine is learned.

Dr. Richards shrinks from producing a dependency problem if everyone is obliged to take two years of postgraduate training. As one who has striven to set up postgraduate teaching programs, I have been impressed by how often the meetings are attended by those who have had more than one year internship.

This type of dependency on continuing medical education might be a good thing to foster.

PAUL H. PFEIFFER, M.D.
Waterville, Maine

From the Secretary's Notebook

Summary of Proceedings, Interim Meeting, M.M.A. House of Delegates, April 11, 1965 at Brunswick, Maine

The interim meeting of the Maine Medical Association House of Delegates was called to order at 2:30 p.m. by the President-elect, John F. Dougherty, M.D., who turned the meeting over to the Speaker of the House, Linus J. Stitham, M.D. There was a total attendance of 62, including 33 delegates, 15 alternates, 10 members of the Council, 3 committee chairmen and the Secretary-Treasurer.

A summary of this meeting has been sent to the President, Secretary and Delegates and Alternates of each County Medical Society and to the Officers and Councilors of the Maine Medical Association. Consequently, only a brief resumé will be published in the Journal.

The Financial Statement of Income and Expenditures for fiscal year ending December 31, 1964 and budget proposed for fiscal year ending December 31, 1966 was presented by the Budget Committee, Drs. Paul S. Hill, Jr. and George E. Sullivan. Final action on the budget proposed for 1966 will take place at the annual meeting of the House of Delegates on June 13 at The Samoset, Rockland, Maine.

The President-elect announced the appointment of the following members to serve on the Nominating Committee: (First District) Charles R. Geer, M.D., Portland — Chairman, (Second District) Edward L. Reeves, M.D., Lewiston, (Third District) Paul A. Fichtner, M.D., Bath, (Fourth District) Edgar J. Smith, M.D., Fairfield, (Fifth District) Russell M. Lane, M.D., Blue Hill, (Sixth District) Arthur N. Lieberman, M.D., Bangor.

The report of the Nominating Committee shall be the First Order of Business at the Second Meeting of the House of Delegates on Sunday, June 13 at 3:00 p.m.

Committee reports were presented by John D. Denison, M.D., Chairman of the Legislative Committee; Richard C. Wadsworth, M.D., Chairman of the Postgraduate Education TV Committee and George W. Bostwick, M.D., Chairman of the Ad Hoc Committee. Dr. Wadsworth's committee suggested "several possible choices for the membership to consider financial support for these education programs. 1. An individual assessment for each member of the society. 2. An increase in the annual dues of the M.M.A. with assignment of specific funds for the continuation of postgraduate education program. 3. No financial support from the membership but reliance upon other organizations to organize and support such programs." It was voted that Dr. Bostwick's committee be requested to bring in definitive recommendations for presentation at the June meeting of the House of Delegates.

The Executive Director, Daniel F. Hanley, M.D., presented an outline of the program for the annual meeting. The Program-in-Brief for this meeting will be found on pages 117 and 118.

Dr. Hanley also discussed malpractice insurance and urged that members carry office liability insurance and malpractice insurance with the same company.

The Maine Cancer Society presented a check for $2,000 to the Maine Medical Education Foundation.

In a brief report relative to the Maine Medical Education Foundation, Dr. Hanley stated that in 1964 grants totaling over $17,000 were awarded to twenty-three medical students from Maine.

The members were reminded that election of Councilors for the following districts will take place at the Second Meeting of the House of Delegates on Sunday, June 13 — Third District (Knox, Lincoln-Sagadahoc Counties), Fourth District (Kennebec, Somerset, Waldo Counties).

George W. Wood, III, M.D., Alternate Delegate to the AMA House of Delegates, presented a brief report of the Special Meeting of the AMA House of Delegates which was held in Chicago in February.

The meeting adjourned at 4:45 p.m.

(Copy of the Summary of Proceedings of this meeting will be sent to any member of the Maine Medical Association on request.)

Announcement

American Cancer Society
1965 Scientific Session

The 1965 scientific session of the American Cancer Society will be held at the Drake Hotel in Philadelphia, Pennsylvania on June 16, 1965.

A symposium on *Hormones and Chemotherapy for Cancer — A Critical Appraisal* is open to all members of the medical and dental professions and students. There is no advance registration or registration fee.

Topics of interest will include: *Nature and Example of Joint Clinical Studies,* Dr. Lyndon Lee, Jr.; *Therapy in Advanced Breast Carcinoma* (Excluding Radiation), Drs. Edward F. Lewison, Michael J. Brennan and B. J. Kennedy; *Melanoma and Adjuvant Therapy,* Drs. Donald B. Rochlin and Warren H. Cole; and *Chemotherapy for Lymphomas and Leukemias,* Dr. Emil Frei, III.

For further information write to: Director of Professional Education, American Cancer Society, 219 East 42nd Street, New York, New York 10017.

Book Review

Bilharziasis, Ciba Foundation Symposium. $11.50.

This Ciba Foundation book presents a summary of recent thought and advances in *Bilharziasis.* Most of the experts are writing from their experiences in Africa, where this parasitic disease still flourishes. Interesting aspects of the immunological features of the disease are discussed. For this part of the world, the book is fortunately of only academic interest.

ANTHONY BETTS, M.D.
Portland, Maine

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The Journal
of the
Maine Medical Association

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Simultaneous Bilateral Acute Angle-Closure Glaucoma

KEVIN HILL, M.D.

Acute angle-closure glaucoma characteristically presents as a *unilateral* ocular inflammation. The affected eye is uncomfortable, vision is blurred, the pupil is semi-dilated and reacts sluggishly or not at all, and the intraocular pressure is palpably increased. The patient may be so overcome by the nausea and vomiting which so often accompanies the acute rise of intraocular pressure that the gastro-intestinal symptoms may, on occasion, overshadow the ocular signs and symptoms. Because the eye cannot long withstand high levels of intraocular pressure without devastating and irreparable damage to the optic nerve, acute angle-closure glaucoma is truly a medical emergency. This acute form of glaucoma has been repeatedly described in the ophthalmic and general medical literature and, happily, is a diagnosis readily suspected by the non-ophthalmic medical practitioner when confronted by a red, painful eye.

Although the standard ophthalmic texts record the fact that both eyes of patients who develop acute angle-closure glaucoma are anatomically pre-disposed to manifest the condition, and it is well-known that an attack in one eye may be followed by a similar affection of the fellow eye, it is not generally appreciated that simultaneous bilateral episodes of acute angle-closure glaucoma may occur. This is not too surprising, since the occurrence of simultaneous bilateral acute angle-closure glaucoma is rare.[1,2,3] In one study of fifty-six cases of acute angle-closure glaucoma, only three presented with bilateral involvement.[4] Nevertheless, we have recently seen two such instances, in both of which the diagnosis was missed by a competent general practitioner. In both instances, the bilaterality of the condition was a misleading factor.

It is the purpose of this report to record instances of the rare occurrence of simultaneous bilateral acute angle-closure glaucoma and to remind our non-ophthalmic colleagues that this dangerous medical emergency can, and does, occur.

CASE REPORTS

CASE 1 – (F.M.) A 73-year-old female was seen in her home because of a complaint of blurred vision and inflammation of both eyes. She had been discharged from the hospital recently with a diagnosis of carcinoma of the breast with metastases to the left pleural cavity (confirmed by thoracentesis). At home, she complained of frontal headache and a solicitous relative applied a liniment* to her forehead. Following this, both eyes of the patient became inflamed and uncomfortable. Her family physician prescribed an antibiotic-steroid ointment** for relief of her ocular symptoms, but they persisted and her vision became progressively worse.

When seen at her home, the patient could see only enough to count fingers at four feet with either eye. There was marked conjunctival and ciliary injection and moderate corneal edema. The pupils were semi-dilated and fixed, the anterior chambers appeared shallow on gross inspection and the ocular tension was 50 mm. Hg (Schiotz) in each eye. Gonioscopic and ophthalmoscopic examinations were unsatisfactory at the time.

Intensive treatment with miotics and acetazolamide was instituted but the ocular tension remained the same during the next three hours. The patient was then hospitalized and given an intravenous infusion of 20% mannitol. The ocular tension promptly fell to normal levels and her vision improved somewhat. Gonioscopy then revealed narrow anterior chamber angles with peripheral anterior synechiae superiorly in both eyes. Ophthalmoscopy was not entirely satisfactory due to nuclear sclerosis of both lenses and the patient's miotic pupils; however, a good red fundus reflex was obtained in all quadrants. The anterior chambers were of moderate and even depth.

The ocular tension was easily maintained at normal levels by miotics and because of the patient's precarious state of health no ocular surgery was done. She was discharged and seen at

*Morning After Rub, T. Noonan, Co.
**Ophthocort®, Parke, Davis & Co.

home where her eyes were comfortable, and ocular tension normal and the vision somewhat improved. However, she soon developed recurrent pleural effusions due to the metastatic carcinoma and died shortly thereafter. Autopsy permission was refused.

CASE 2 – (E.A.) A 76-year-old white female was seen by her family physician for pain and injection of her left eye. A diagnosis of conjunctivitis was made and an antibiotic-steroid ointment* was prescribed. The injection and pain diminished markedly during the next week but then recurred six days later. Another antibiotic-steroid ointment** was prescribed but her symptoms did not clear. The right eye became similarly affected two days later and the vision became blurred. She was then referred for further evaluation.

Vision with glasses was reduced in both eyes to ability to count fingers. Both corneas were "steamy," the anterior chambers were shallow and the pupils were dilated. The ocular tension was 85 mm. Hg (Schiotz) in both eyes. Gonioscopic examination of the anterior chamber angles was not satisfactory initially due to the corneal edema. Intensive treatment with miotics and acetazolamide lowered the ocular tension after two hours to, O.D. 15, and O.S. 60 mm. Hg (Schiotz). The corneal edema was somewhat less and goniscopy then revealed the following: O.D., angle narrow but open temporally and inferiorly for approximately 180 degrees. Elsewhere, the angle appeared closed. O.S., angle closed except for a small cleft temporally.

The patient was hospitalized and treated with miotics and local steroids to reduce the considerable ocular inflammation which she had. The ocular tension was normalized and remained so until bilateral periphal iridectomies were performed five days later. The post-operative course was uneventful and the ocular tension has remained normal in both eyes without treatment. A gratifying development was the improvement of vision to 20/30 in the right eye and 20/25 in the left eye. There are a few small lens opacities (glaucomaflecken) in the right eye but the discs appear normal.

Comment

There are several features of these two case reports which are worthy of comment. In Case 1, it is not clear what role, if any, the liniment played in the genesis of the acute angle-closure glaucoma. The patient had a frontal headache to begin with, and this may have been the first sign of increased intraocular pressure. The liniment consists mainly of alcohol and menthol and does not contain any specific mydriatic ingredients. It most probably caused a chemical irritation of the cornea and conjunctiva but whether or not it caused intraocular congestion sufficient to close the anterior chamber angles is speculative. In case 2, the initial episode of inflammation of the left eye may have been conjunctivitis. However, the fact that the inflammation cleared with the topical administration of antibiotic and corticosteroid does not exclude the possibility that this was angle-closure glaucoma for it is well known that such attacks may terminate spontaneously. If the first episode *was* conjunctivitis it only served to confuse the diagnostic picture when bilateral ocular inflammation developed a week later for it is not uncommon for conjunctivitis to spread from one eye to the other.

*Neomedrol®, Upjohn Co.

**NeoDecadron®, Merck, Sharp & Dohme

TABLE I

Differential Diagnostic Features of Conjunctivitis and Acute Angle-Closure Glaucoma

	Conjunctivitis	*Angle-Closure Glaucoma*
Pain	Sandy, burning	Severe, V Cranial Nerve
Vision	Good, or Blur cleared by blinking	Blurred, Halos around lights
Secretion	Mucopurulent	Watery
Injection	Superficial	Ciliary flush
Pupil	Normal	Semi-Dilated
Cornea	Clear	"Steamy"
Ocular Tension	Normal	High
Systemic Sx.	None	Nausea, Vomiting

N.B. The gradations of severity of these signs and symptoms are such that the two conditions are sometimes not easily differentiated. An awareness of the above differential features will lead to the correct diagnosis in the great majority of cases.

Indeed, if acute angle-closure glaucoma is misdiagnosed it is usually thought to be conjunctivitis. The differential diagnosis of these two conditions (Table I) is well known but it is well to emphasize that palpation of a red eye through the gently closed lids will often prove to be the most help in differentiating the two conditions. The firmness of the globe with unilateral acute angle-closure glaucoma usually is in striking contrast to the normal fellow eye. In simultaneous bilateral cases of angle-closure glaucoma, one has to rely upon a sense of what is a normal palpable ocular tension, and this can best be acquired through practice – on normal eyes as well as inflamed eyes.

Finally, a word should be said about an important aid to the diagnosis of acute angle-closure glaucoma, namely, a high index of suspicion. If a patient with an inflamed eye has little or no mucopurulent discharge, or has a palpable difference in ocular tension, or fails to respond promptly to topical antibiotics, or has accompanying systemic signs such as headache, nausea or vomiting, the ocular tension should be taken with a tonometer and an ophthalmologic consultation should be obtained.

The present report emphasizes one more facet of this dangerous ocular disease that is of interest to the physician with the high index of suspicion. Acute angle-closure glaucoma can present as a simultaneously bilateral affliction.

Acknowledgment: I am indebted to Dr. H. F. Hill for permission to report Case 2.

References

1. Chandler, P. A.: Personal Communication, December, 1964.
2. Duke-Elder, S.: Textbook of Ophthalmology, V.3, C. V. Mosby Co., St. Louis, 1947, p. 3366.
3. Hill, H. F.: Personal Communication, January, 1965.
4. Hill, K.: Unpublished data.

33 College Avenue, Waterville, Maine

Human Infestation With Canine Scabies

JAMES F. MADISON, M.D.

The mite of human scabies, *Sarcoptes scabiei var. hominis,* causes a characteristic eruption when it infests the skin. The primary lesions are red papules and burrows with excoriations and impetigo as secondary changes. The sites of predilection are the interdigital webs of the fingers, the flexor surfaces of the wrists, the elbows, anterior axillary folds, nipples, belt line, buttocks, and male genitalia. Itching may be severe and is often nocturnal. The eruption appears after an incubation period which may last several weeks during which the patient becomes sensitized to the mite or its excretions. Since the mites reproduce on man, the infestation may persist indefinitely unless a scabicide is used.

The mites of animal scabies occasionally attack man, but the diagnosis may be more elusive because the eruption is of varying morphology.[1–4] Red macules, papulovesicles, and wheals on the covered portion of the body are the usual findings. There are no burrows as in human scabies. The incubation period is brief, so that the lesions appear within a few hours to three days after exposure. Animal mites do not propagate on human skin. Consequently, the disorder is self-limited providing the source of contact is eliminated.

If the diagnosis of animal scabies is suspected, it can often be confirmed by finding the mite in scrapings from the tops of the lesions. The mites of animal scabies look just like the mite of human scabies, but apparently there are biologic variations since each variety of *S. scabiei* is able to reproduce only on its particular host. Animal mites infest swine, cattle, horses, mules, sheep, goats, dogs, cats, and various wild animals. The following is a report of two human infestations with *Sarcoptes scabiei var. canis.*

Case Reports

Case 1 – A 61-year-old widow developed a rash under the breasts four weeks before consultation. The eruption spread over the abdomen, hips, buttocks and arms as small papules surmounted by central vesicles. Some papules had a telangiectatic base measuring up to 1.5 cm. in diameter. The lesions were discrete but grouped. There were no burrows. Itching was paroxysmal, being worse during the day, and prompted such vigorous scratching that bruises were produced.

On the first office visit scrapings from several lesions were examined for mites, but none were found. Nevertheless, the patient was instructed to apply a scabecide, crotamiton,* from the chin down and repeat the application in 24 hours. One week later the original lesions had healed without scar. However, new ones were present in the same distribution. Biopsy of a fresh papule showed acute dermatitis, but no mite was found in the stratum corneum. Subsequent scraping finally revealed two mites, morphologically identical to *Sarcoptes scabiei, var. hominis.*

A veterinarian then was asked to examine the patient's dog for mites and was able to demonstrate them about the animal's ears. The dog had been thought to have an allergy because of excessive scratching. It lived in the home, often sleeping on chairs and on the patient's bed. The dog was treated with a scabecide and the patient applied crotamiton to herself once again. The lesions healed and this time no more mite bites appeared.

A frequent visitor in the patient's home was said to have a similar eruption which cleared spontaneously.

Case 2 – A 41-year-old patient and two other adult members of her household developed an itchy eruption on the covered portions of the body soon after purchasing a dog in June, 1964. The dog had scaly skin about its haunches which it scratched repeatedly. The patient's occupation kept her away from home Monday through Friday each week. She noticed that pruritus and rash decreased during the week only to become worse each weekend. Examination revealed an eruption similar to that in Case 1. I was unable to demonstrate mites in the lesion, nor did the veterinarian find them on the dog. Nevertheless, antiscabetic treatment of both dog and patient was followed by healing of the involved skin.

Summary

This report is presented as a reminder that the mites of animal scabies can and do infest man. The ensuing eruption is variable, but the diagnosis can often be established by demonstrating mites in the lesions. Treatment of both patient and the animal source with antiscabetic preparations produces a cure.

References

1. Pillsbury, D. M., Shelley, W. B., and Kligman, A. M.: Dermatology, Philadelphia: W. B. Saunders Company, 1959, p. 1055.
2. Schwartz, L., Tulipan, L., and Birmingham, D. J.: Occupational Diseases of the Skin, ed 3, Philadelphia: Lea and Febiger, 1957, p 674.
3. Emde, R. N.: Sarcoptic Mange in the Human, Arch. Derm. 84:633, 1961.
4. Beck, A. L. Jr.: Animal Scabies Affecting Man, Arch Derm. 91:54, 1965.

*Eurax, Geigy Pharmaceuticals, Ardsley, N. Y.

60 Colchester Avenue, Burlington, Vermont

The Ordeal of John Fairfield: A Tragedy Without Footnotes

S. M. Rabson, M.D.

John Fairfield: born 1797; Member of House of Representatives, 1835-1837; Governor of Maine, 1839-1840, 1842-1843; United States Senator, 1843-1847; while in Washington, he wrote frequently to his wife who remained at home.

Dec. 15th, 1846. "On calling at the 6th Auditor's office, Mr. Washington's, he inquired particularly about my knees . . . and said he had a clerk in the office who . . . had just such a knee which was entirely cured by a Dr. McGruder of this city. He sent for the clerk who confirmed all that Washington had told me. Both pressed me hard to let him operate on me . . . being sure . . . he could cure me. . . . I took some pains to find out who and what this Dr. McGruder was. The result is that he is regarded here as exceedingly skillful physician and surgeon, is rather rough and odd, but a kind-hearted man, and one in whom his employers have great confidence. I learn also that he is physician for Pakenham, the British minister . . . the Russian minister . . . the French minister. . . . Today I called on him. I liked his looks very much. He is no dandy, but has a broad, good-natured Scotch face and looks like a man of genius. . . . This afternoon at 4 o'clock he called, examined, etc., and says the case is one of dropsy and that he can cure it. I told you the other day that they had not been better for years. Strange to say, the next day they were hardly ever worse and now my legs and ankles are much swollen. He says the dropsy is beginning to spread and will soon extend upward to the abdomen unless something is done . . . would merely make an incision and draw off the water. He says I shall not be confined more than a week . . . there is not the least danger of producing a stiff joint or doing any other injury."

Dec. 16th, 1846. "My health, I think, continues improving, except my lameness. That is just now very bad. The swelling extends to my ankles and feet, and compelled me to buy some thin merino socks."

Dec. 19th, 1846. "Dr. McGruder has just been here agreeably to his appointment and has designated Tuesday next at 11 o'clock to perform his operation upon my knees. . . ."

Dec. 22nd, 1846. " 'The long agony is over,' . . . My knees have been operated upon and so far as we can now judge with the most perfect success. . . . When the Doctor got ready to put in his knife, 'Now,' said he, 'if I am right in my opinions of your case a yellow gelatinous fluid will follow the knife.' He then inserted the instrument on the outside of the knee joint and just above it and his prediction was fully verified. From both knees there was drawn about a pint and a half of yellow fluid, nearly the consistency of white of egg, not quite so thick. He says that at least a pint more will yet be drawn off. He then bandaged my knee and leg below it tightly and left me. . . . Cousins Moses Titcomb and Richard Cutts were present at the operation. . . . Richard pressed me to call in Dr. Hall as advising physician, but I declined, having full confidence in McGruder's honest face, strong good sense, his simplicity and want of pretention, and apparent frankness and honesty. . . ."

Dec. 23rd, 1846. "The Doctor has just left me. He opened my knees again, in new places, and drew off about another pint of blood and matter and poked pieces of cloth into the orifices to keep them open. He hurt me much more today . . . and the irritation caused by the pieces of cloth in the holes is by no means agreeable, I can tell you. He postponed the operation with the Port wine until tomorrow. As I understand him, the coming away of the wine will be accompanied by the healing fluids and the knees will then be in the proper condition to heal, or rather for the parts so long separated to come together again. . . ."

Dec. 24th, 1846. "This morning the Doctor opened my right leg on the inside but got nothing but blood. He then with a syringe forced cold water into it and afterward Port wine, both of which were immediately ejected, and the leg bound up. The operation this morning was very severe and my leg is yet, say 3 o'clock, in a good deal of pain. To the left leg he did nothing more, believing that there had been already inflammation enough. . . ."

Dec. 25th, 1846. "Yesterday the Doctor made a new opening which brought on my old neuralgic pain which lasted all day. In the evening, by direction of the Doctor, I enveloped one knee with a bread and milk poultice. This relieved the pain, and was followed by one of the best night's sleep I have had since leaving home. . . . The doctor has just been in, but has done nothing, says everything is working to his satisfaction. . . . I walk my chamber a good deal, by direction, for the purpose of producing irritation and consequent inflammation, – the very thing that most other surgeons seem to dread. . ."

Dec. 26th, 1846. "The doctor came. . . . The opening in the left knee I found very sore and beginning to fester. On opening it, however, we found that the piece of cotton cloth had never been extracted. . . ."

Dec. 27th, 1846. "The inflammation has nearly ceased and the healing process appears to be going on

gradually and healthily. The knees, to be sure, are still swollen – but the swelling is mainly, I apprehend, in consequence of the incisions, or the sores made by them. It will probably be months before an entire restoration to the original strength of the joints, – though for most purposes I may be considered well. . . ."

Dec. 29th, 1846. "Yesterday, Monday, one day within the week set by the Doctor, I went to the Senate Chamber. The joints are now considerably swollen and bandaged tightly, I suppose to reduce the swelling and to aid the healing process. . . ."

Jan. 1st, 1847. "I could not undertake to crowd my way through the throng at the President's [New Year's reception] with my game legs. . . . Of my lameness I am obliged to say the joints are still weak, and it is with some difficulty that I can walk a considerable distance. . . ."

Jan. 10th, 1847. "Bulletin No. 40, – save one. Knees growing better. Have not seen the Doctor since a week last Thursday and don't know what has become of him."

Jan. 28th, 1847. "I cannot close without alluding to my knees, and I am happy to say that the last two days have been much better than they have been since the operation, while the two days before they were worse. If things go on as favorably as they have for the last two days, you and I will dance a jig when we have the pleasure of meeting. . . ."

There were no further references in the next letters, the last, Feb. 24th, 1847. A new series was dated December, 1847.

Dec. 16th, 1847. "Strange that I have not seen Dr. Mcgruder yet. . . ."

Dec. 19th, 1847. No reference to knees.

Dec. 23rd, 1847. "Dr. Mcgruder called to see me today for the first time. His confidence in his ability to cure me is not only undiminished but strengthened by the success of other cases which he has had. So it is all arranged. He is to be here tomorrow morning and operate upon my knees. He is unwilling that I should inhale ether and that project is abandoned. He says that addition to his prejudices against it, that in my case it would do no good, inasmuch as the vitriol is to be kept in the cavities of the knee twelve hours or more, and that the pain will be more severe at the close than at the commencement. Oh dear! What a prospect I have before me. The fire burning in my flesh for 12 hours! It is hard to think of, but I submit willingly under the probable chance of cure. Cousin Moses and Mr. Hammons are to be here. I did intend to send this letter by this night's mail, but not wishing to give you 24 hours of anxiety about me, I think I will keep it until after the operation and then finish it, so that you will have the annunciation and result together. So no more tonight." The letter was never completed.

It was Gideon Welles who finished the letter in his diary. At the time, he served as civilian head of the Navy Department's Bureau of Clothing and Provisions; he was later to be Lincoln's Secretary of the Navy.

"John Fairfield, Senator from Maine died on the evening of 24th Decr. 1847. On the morning of that day he was in good health. During the day he submitted to a surgical operation on both of his knees, which were lame, and which for years had given him trouble. He had consulted eminent surgeons as to obtaining relief who had strongly remonstrated against it, as likely to be attended with no permanent relief, and which would be attended with great risk. Being a man of great physical courage the latter admonition was lost upon him and having found a Dr. Mcgruder of this city who claimed that he could effect a cure he last winter submitted to an operation. The water which had gathered in his joints was drawn off and infusion of port wine made. For some months he found his knees better, but the complaint having returned he determined to submit to a more thorough experiment. Between twelve M. and one on the 24th, being in excellent health of spirits the physician made an incision, drew off the water and infused sulphate of copper which he bound in and then left his patient promising to return in half an hour. But an hour passed of most excruciating agony, and at last Gov. F. sent his attendant for Dr. M. It was instead of half an hour, an hour & a half before he returned. In the meantime, the poison had done its work a general paralysis had set in. Gov. F. said he could not raise his arm – that he found it difficult to speak. On his appearance the Dr. manifested great indifference administered an opiate and again left. The Governor apparently aware of what would be the termination, turned his head upon the pillow remarked 'This is melancholy' and never spoke again. He died a little before eight, about seven hours after the infusion of the sulphate of copper. . . . Had he professed less physical courage, he would not have been the victim of a reckless experiment. . . . The traits of his character and his goodness of heart made me like him. In addition to this he had been my warm and earnest friend, when others under higher obligations to me failed a friendly part."

References

1. Staples, A. G. (editor): The Letters of John Fairfield. Lewiston Journal Company, Lewiston, Maine, 1922.
2. Welles, Gideon: Unpublished diaries. Huntington Library, San Marino, California.

2264 West Live Oak Drive, Los Angeles, California 90028

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Phenylketonuria Detection

CHARLES H. OKEY, Ph.D.*

Authority to require testing of all newborn infants for metabolic defects leading to mental retardation has been given to the Department of Health and Welfare by the 102nd Legislature. The law will become effective ninety days after the legislature adjourns. By that time, the Department will make available its Rules and Regulations implementing the law. It is anticipated the regulation will call for phenylketonuria screening only. If and when newer knowledge indicates the desirability of testing for other metabolic defects, the specifications will be revised to include additional tests.

Because of the new law and because of recent and anticipated changes in the PKU program as originally conducted, it was felt advisable to communicate this information and to summarize the current status of PKU as widely as possible within the medical community. One major change in the program stems from a concern over the fact that only about 70% of all infants are being screened for PKU despite the ready availability of the test in the Diagnostic Laboratory. Reasons for this are not known but it would appear that perhaps early discharge from the hospital, before the infant had protein intake sufficient to challenge its ability to metabolize phenylalanine, might be the basis for not sampling these infants' blood. Also, discharge at times when personnel responsible for blood sampling were not on duty might account for the lower yield of specimens.

To try to increase the number of children tested and to increase the sensitivity of the screening, it was decided to allow for a follow-up test at 4 to 6 weeks of age by providing hospitals with a two-part kit. One portion of the kit is to be used in the hospital and the other portion given to the mother for a second specimen to be taken in the physician's office at the time of the baby's first visit to the doctor. In addition to the filter paper for the blood sample, a sterile lancet and an envelope for mailing are included in the follow-up kit. Specimens taken in the hospital and in the doctor's office are forwarded to the Diagnostic Laboratory for testing.

Testing in the Diagnostic Laboratory is carried out by the use of the bacterial inhibition assay method of Dr. Robert Guthrie, Department of Pediatrics, School of Medicine, State University of New York at Buffalo. The test is a sensitive procedure indicating phenylalanine levels through a range of one to twenty mgm % or greater at intervals of 1 to 2 mgm. It is not entirely specific in that other elevations of blood amino acids such as leucine, tyrosine and methionine can produce results similar to phenylalanine. This lack of specificity makes it necessary to use some more definitive procedure before a diagnosis is made. A fluorometric test appears at the present time to be the method of choice although the LaDu test or a chromatographic analysis may be used with some confidence if adequate controls are employed.

The national evaluation of the Guthrie test in 1962-63 was based on a sample of 400,000 newborn infants in the United States with tests carried out in the laboratories of several state health departments. Maine participated in the evaluation and tested 10,000 infants. From the entire sample, 39 confirmed cases of PKU were identified, reflecting a frequency of 1 per 10,000 births, a figure double that thought to obtain prior to the study. Sixty-five % of the infants were tested at 4 to 6 weeks of age by a follow-up urine test without adding any additional cases. False presumptive positives occurred at the rate of 1 per 1500 when the bloods were screened at a 6 mgm % level. Screening at a lower value of 4 mgm %, the rate is about 1 per 300 infants which means about 30 infants would be subjected to a repeat test for each case of PKU. Screening at the 4 mgm % is being followed at the present time because of the inherently greater sensitivity. Thirty thousand specimens have been tested in the Diagnostic Laboratory up to the present time. Not a single confirmed case of PKU has been found among the infants tested. During the time tests have been

Continued on Page 146

*Director, Diagnostic Laboratory

Maine Medical Association

Program

112th Annual Session

June 13, 14, 15

THE SAMOSET ROCKLAND, MAINE

Program

Arranged by the Scientific Committee

ALBERT L. HUNTER, M.D., Camden
Chairman

RICHARD P. LANEY, M.D., Skowhegan

CLEMENT A. HIEBERT, M.D., Portland



Dr. Hunter

Sponsors:

The Scientific Program for the annual session of the Maine Medical Association is made possible this year by grants from

Merck Sharp & Dohme Postgraduate Program

Eli Lilly and Company State Medical Convention Program

Geigy Pharmaceuticals

Maine Cancer Society

The Maine Heart Association, Inc.

Pine Tree Chapter, The Arthritis and Rheumatism Foundation

in cooperation with the Technical Exhibitors and the Council of the Maine Medical Association.

For this cooperation and support the members of the Scientific Committee are grateful.

Albert L. Hunter, M.D., Chairman

Information

Registration:

Registration throughout the session will be in the Lobby at The Samoset. Registration fee $1.00.

Sunday	June 13 — 9:00 A.M. to 5:30 P.M.
Monday	June 14 — 9:00 A.M. to 6:00 P.M.
Tuesday	June 15 — 9:00 A.M. to 4:30 P.M.

Telephone: The number at The Samoset is Rockland, 594-8411.

Visiting Delegates:

Introduction of Visiting Delegates will take place at meetings of the House of Delegates on Sunday, June 13 or at the General Assembly, Monday afternoon, June 14.

Special Exhibits:

These exhibits which are listed on page 143, will be located in the Ballroom.

Technical Exhibits:

This year forty-five companies are contributing to the success of the annual session program by participating in the Technical Exhibits. You will pass these exhibits on your way through the lobby en route to the Ballroom (where the Scientific Sessions will be held) to the Dining Room or to the Golf Course.

Stop and thank them for their assistance in making this program possible. A list of the exhibiting companies and representatives will be found on pages 144 through 145 of this program.

Sunday, June 13

9:30 A.M. First Meeting of the House of Delegates

Call to order: JOHN F. DOUGHERTY, M.D., President-Elect

Presiding: Speaker of the House, LINUS J. STITHAM, M.D.

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

5:00 P.M. **Visit the Technical Exhibits**

6:30 P.M. Dinner

Speaker: MR. STEVE MERRILL, Brunswick, Maine

Subject: **Maine Yarn Spinning**

Steve Merrill comes from Skowhegan, Maine and is a writer and photographer as well as a story teller. His work has appeared in national magazines, and his daily column, "SAND 'N' SALT" is a regular feature of Maine's leading newspaper.

Monday, June 14

Scientific Program

9:45 A.M. to 12:00 NOON

Presiding—ALBERT L. HUNTER, M.D.

9:45 A.M. **Some Clues in the Care of Cardiacs**

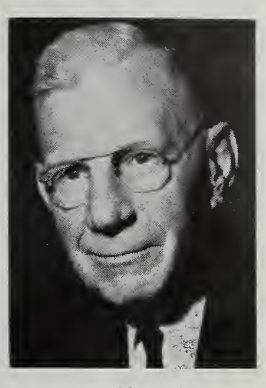
SAMUEL A. LEVINE, M.D., Consultant in Cardiology, Peter Bent Brigham Hospital, Brookline, Massachusetts

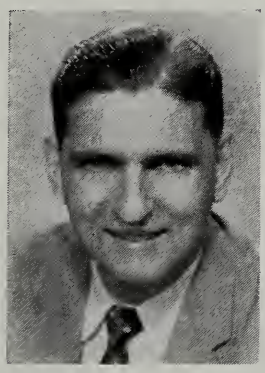
Sponsored in part by a grant from Merck Sharp & Dohme Postgraduate Program and The Maine Heart Association, Inc.


Mr. Merrill


Dr. Levine


Dr. Crile


Dr. Kennedy


Dr. Hannigan


Dr. Lincoln


Dr. Woodcock

11:00 A.M. **The Role of Simple Mastectomy in the Treatment of Cancer of the Breast**

GEORGE CRILE, JR., M.D., Head of the Department of General Surgery, Cleveland Clinic Foundation, Cleveland, Ohio

Sponsored in part by a grant from Merck Sharp & Dohme Postgraduate Program and the Maine Cancer Society.

12:00 NOON to 2:00 P.M. Luncheon

Scientific Program

1:30 to 4:00 P.M.

Presiding—RICHARD P. LANEY, M.D.

Sponsored by the Maine Chapter, American College of Surgeons and Maine Trauma Committee

1:30 P.M. Business Meeting, Maine Chapter, American College of Surgeons

2:00 P.M. **Controversial Questions in the Management of Surgical Problems**
(An Informal Discussion)

GEORGE CRILE, JR., M.D., Cleveland, Ohio

3:00 P.M. Panel – **Problems of a Case of Multiple Trauma**

Moderator: ROBERT H. KENNEDY, M.D., Director of Field Program, Committee on Trauma, American College of Surgeons, New York

Panel:

CHARLES A. HANNIGAN, M.D., Internist, Auburn

H. RICHARD HORNBERGER, M.D., Surgeon, Waterville

JOHN R. LINCOLN, M.D., Anesthesiologist, Portland

JOHN A. WOODCOCK, M.D., Orthopedist, Bangor

4:00 P.M. **General Assembly**

Presiding, THOMAS A. MARTIN, M.D., President

Election of President-Elect

6:00 to 7:00 P.M. Social Hour, Dutch Treat, Ballroom

7:00 P.M. Annual Banquet

Presentation of Honorary Pins

Speaker: J. M. HAYMAN, JR., M.D., Dean, Tufts University School of Medicine, Boston, Massachusetts

Subject: **Trends in Medical Education**

Dr. Hayman, Dean and Professor of Medicine at Tufts University School of Medicine and Senior Physician, New England Center Hospital since 1953, is the author of 79 publications on clinical medicine, renal physiology and tropical disease.



Dr. Hayman

Tuesday, June 15

Scientific Program

9:30 A.M. to 12:00 NOON

Presiding—RICHARD P. LANEY, M.D.

9:30 A.M. **The Medical Treatment of Rheumatoid Arthritis**

KENNETH FREMONT-SMITH, M.D., Junior Associate in Medicine, Peter Bent Brigham Hospital, Brookline, Massachusetts

Sponsored in part by a grant from Merck Sharp & Dohme Postgraduate Program, and the Pine Tree Chapter, The Arthritis and Rheumatism Foundation.



Dr. Gallagher

10:30 A.M. Panel – **Your Children and Mine, Their Problems From 12 to 20**

Moderator: J. ROSWELL GALLAGHER, M.D., Chief, The Adolescents' Unit, The Children's Hospital Medical Center, Boston, Massachusetts

Panel:

ACHSA M. BEAN, M.D., Owl's Head

WILLIAM E. SCHUMACHER, M.D., Augusta

Sponsored in part by a grant from Geigy Pharmaceuticals and Eli Lilly and Company State Medical Convention Program.

12:00 NOON to 2:00 P.M. Luncheon



Dr. Schumacher

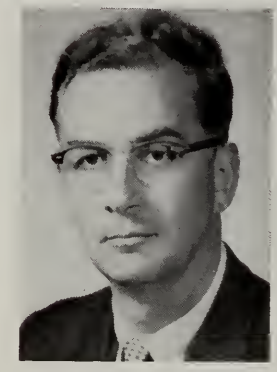
Scientific Program

2:00 to 4:00 P.M.

Presiding—ALBERT L. HUNTER, M.D.

Sponsored by the Maine Medico-Legal Society

2:00 P.M. **Psychiatry and Criminal Responsibility**
PETER W. BOWMAN, M.D., Superintendent, Pineland Hospital and Training Center, Pownal



Dr. Bowman

3:00 P.M. **The Attorney General's Office and the Investigation of Crimes in Maine**

The Honorable RICHARD J. DUBORD, Attorney General of the State of Maine

6:30 P.M. Clam Bake

Presentation of Golf Prizes by

DANIEL R. SHIELDS, M.D., Lewiston, Chairman

Golf Tournament

Specialty Group Meetings

Monday, June 14

2:00 to 4:00 P.M.

Maine Thoracic Society and Maine Society of Internal Medicine

2:00 P.M. Maine Society of Internal Medicine

CHARLES W. STEELE, M.D., Lewiston, presiding

Medical Trends Today – Your Masters or Your Servants?

FRANK P. FOSTER, M.D., Member, Board of Trustees, American Society of Internal Medicine, Boston, Massachusetts



Dr. Foster



Dr. Dulfano

2:30 P.M. Maine Thoracic Society

RUDOLPH HAAS, M.D., Lewiston, presiding

Guide Lines For Treatment of Acute Respiratory Insufficiencies in Chronic Pulmonary Patients

MAURICIO J. DULFANO, M.D., Assistant Professor of Medicine, Tufts University School of Medicine, Boston, Massachusetts

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Maine Society of Clinical Hypnosis

CARL M. HAAS, M.D., Biddeford, presiding

Hypnotic Technics in Therapy

CALVERT STEIN, M.D., American Board of Psychiatry and Neurology, Springfield, Massachusetts

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Maine Society of Obstetrics and Gynecology

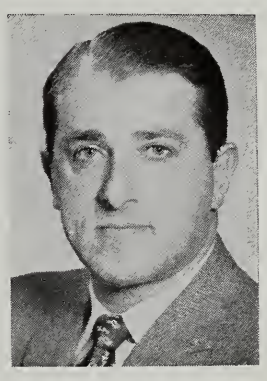
KENNETH W. SEWALL, M.D., Waterville, presiding

The Intra-Uterine Treatment of Fetal Blood Incompatibilities

IRVING UMANSKY, M.D., Chief of Blood Grouping Laboratory, The Children's Hospital Medical Center, Boston, Massachusetts



Dr. Stein



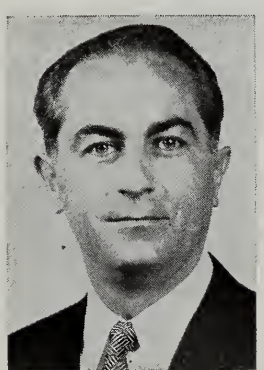
Dr. Umansky

Maine Society of Anesthesiology

FRANCIS X. MACK, M.D., Portland, presiding

The Anesthesiologist and Hemorrhagic Shock

C. PAUL BOYAN, M.D., Chief of Anesthesiology Department, New York Sloan-Kettering Cancer Center, New York



Dr. Boyan



Dr. Montgomery

Tuesday, June 15

10:00 A.M. Maine Medico-Legal Society

Business Meeting

IRVING I. GOODOF, M.D., Waterville, presiding

2:00 to 4:00 P.M.

Ear, Nose and Throat Group

JOHN E. WHITWORTH, M.D., Bangor, presiding

Osteoplastic Frontal Sinus Operation

WILLIAM W. MONTGOMERY, M.D., Associate Professor, Harvard Medical School, Boston, Massachusetts

Section on Ophthalmology

RALPH A. GOODWIN, JR., M.D., presiding

Report of Eye Clinics Visited From Japan to Yugoslavia (with slides)

HOWARD F. HILL, M.D., Waterville

Panel Discussion: Crede Method of Prophylaxis in the Newborn

Moderator: OTIS B. TIBBETTS, M.D., Lewiston

Panel:

DEXTER J. CLOUGH, 2nd., M.D. Bangor

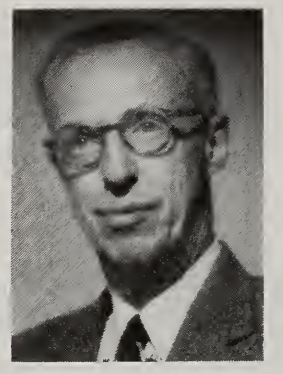
RICHARD H. DENNIS, M.D., Waterville

RICHARD J. GODUTI, M.D., Portland

JOU S. TCHAO, M.D., Lewiston

Report of the Howe Laboratory Memorial Fund for Merrill J. King, Sr., M.D.

KEVIN HILL, M.D., Waterville



Dr. Hill

Maine Chapter of the American Academy of Pediatrics

Annual Meeting

RUSSELL A. MORISSETTE, M.D., Lewiston, presiding

Luncheon Meetings

12:30 P.M.

Tuesday, June 15

Maine Chapter, American Academy of General Practice

GEORGE W. BOSTWICK, M.D., Newcastle, presiding

Huddilston Medal Award Committee

Ear, Nose and Throat Group

Amy W. Pinkham Fund Committee

SPECIAL NOTICES

Election of President-Elect

The Election of a President-Elect will take place at the General Assembly, June 14 at 4:00 p.m.

Election of Councilors

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 13 at 3:00 p.m.

Third District – Knox and Lincoln-Sagadahoc

Fourth District – Kennebec, Somerset and Waldo

In accordance with the By-Laws, "Nominations for members of the Council for any District where there is a vacancy shall be made by a caucus of the members of the House of Delegates of that District. Each candidate for Councilor must be a resident of the District for which he is nominated."

Council Meetings

The Council will meet on Saturday, June 12 and daily throughout the session at a time and place to be announced.

Tufts Medical Alumni Reunion

Monday, June 14, 6:00 to 7:00 p.m.

Committee Meetings

Committee on Recruitment, Aid and Placement Monday, June 14 – 4:30 p.m.

Health Insurance Committee Breakfast Meeting Tuesday, June 15 – 8:00 a.m.

Golf Tournament

Daniel R. Shields, M.D., Chairman

HONORARY PINS

Presentation of the Association's Honorary Pins will be made by Thomas A. Martin, M.D., President of the M.M.A., at the Annual Banquet, Monday evening, June 14 at 7:00 P.M.

FIFTY-YEAR PINS

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1915:

Cumberland County

Elton R. Blaisdell, M.D., Portland
Bowdoin Medical School

Horace K. Sowles, M.D., Falmouth
Harvard Medical School

Langdon T. Thaxter, M.D., Portland
Harvard Medical School

Kennebec County

Albert S. Crawford, M.D., East Blue Hill
Cornell University College of Medicine

Knox County

Harold M. Frost, M.D., Friendship
Harvard Medical School

Penobscot County

Allan Woodcock, M.D., Bangor
Bowdoin Medical School

Washington County

Charles M. Armstrong, M.D., Robbinston
Tufts University School of Medicine

York County

Carl G. Dennett, M.D., Saco
Bowdoin Medical School

Charles W. Kinghorn, M.D., Kittery
Bowdoin Medical School

Harold W. Stevens, M.D., Saco
Harvard Medical School (1914)

FIFTY-FIVE-YEAR PINS

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Pins in 1960:

Kennebec County

Samuel H. Kagan, M.D., Augusta
Tufts University School of Medicine

Penobscot County

Thomas A. Devan, M.D., St. Petersburg, Florida
Johns Hopkins University School of Medicine

SIXTY-YEAR PINS

Sixty-Year Pins will be presented to the following members who received their Fifty-Year Pins in 1955:

Androscoggin County

Daniel F. D. Russell, M.D., Leeds
Bowdoin Medical School

Cumberland County

Nathaniel B. T. Barker, M.D., Yarmouth
Bowdoin Medical School

Piscataquis County

Fred J. Pritham, M.D., Greenville Junction
Bowdoin Medical School

Visiting Delegates

The Connecticut State Medical Society
CHARLES M. BARBOUR, M.D., West Hartford
SYDNEY LURIA, M.D., Bridgeport

The Massachusetts Medical Society
LAURENCE B. ELLIS, M.D., Boston

Medical Society of the State of New York
GEORGE A. BURGIN, M.D., Little Falls

Vermont State Medical Society
B. ALBERT RING, M.D., Burlington

Delegates to Out-of-State Meetings

The Connecticut State Medical Society
ROBERT M. KNOWLES, M.D., Portland

The Massachusetts Medical Society
HARLAND G. TURNER, M.D., Norridgewock

The New Hampshire and Vermont State Medical Societies
CHARLES D. MCEVOY, JR., M.D., Bangor

Medical Society of the State of New York
ERNEST W. STEIN, M.D., Pittsfield

The Rhode Island Medical Society
LEONARD G. MIRAGLIUOLO, M.D., Bangor

County Delegates

FIRST DISTRICT

Cumberland County Medical Society

Delegates: Stanley B. Sylvester, M.D., 1377 Washington Ave., Portland, Secretary
(2 years)
Charles R. Geer, M.D., 208 Vaughan St., Portland
Howard P. Sawyer, Jr., M.D., 22 Bramhall St., Portland
Maurice Van Lonkhuyzen, M.D., 131 State St., Portland
Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland
David K. Lovely, M.D., 46 Deering St., Portland
Clement A. Hiebert, M.D., 18 Bramhall St., Portland
(1 year)
Robinson L. Bidwell, M.D., 31 Bramhall St., Portland
John F. Gibbons, M.D., 22 Bramhall St., Portland
Charles R. Glassmire, M.D., 37 Deering St., Portland
Donald P. Cole, M.D., 45 Deering St., Portland

Alternates

(2 years)
Hugh P. Robinson, M.D., 27 Deering St., Portland
Raphael F. Turgeon, M.D., 367 Main St., Westbrook
Harold N. Burnham, M.D., 130 Main St., Gorham
Alfred Hurwitz, M.D., 32 Deering St., Portland
Irving J. Poliner, M.D., 235 State St., Portland
Louis A. Ciampi, M.D., 326 Stevens Ave., Portland
(1 year)
Lloyd G. Davies, M.D., 78 Main St., Fryeburg
Stanley W. Kent, M.D., 42 Deering St., Portland
A. Dewey Richards, M.D., 11 Gage St., Bridgton
George F. Sager, M.D., 18 Bramhall St., Portland

York County Medical Society

Delegates: Charles W. Kinghorn, M.D., 4 Wentworth St., Kittery, Secretary
Carl E. Richards, M.D., 34 Winter St., Sanford
Marcel P. Houle, M.D., 200 Alfred St., Biddeford
Robert F. Ficker, M.D., Maine St., Kennebunkport

Alternates

G. Patrick Shaw, M.D., 357 Elm St., Biddeford
Stephen A. Cobb, M.D., 34 Winter St., Sanford
Kenneth E. Leigh, M.D., Brixham Rd., York

SECOND DISTRICT

Androscoggin County Medical Association

Delegates: Donald L. Anderson, M.D., 369 Main St., Lewiston, Secretary
Robert D. Wakefield, M.D., St. Mary's General Hospital, Lewiston
George B. O'Connell, M.D., 11 Lisbon St., Lewiston
Edward L. Reeves, M.D., 179 Sabattus St., Lewiston
Morris E. Goldman, M.D., 185 Webster St., Lewiston

Alternates

Henry C. Thacher, M.D., 117 Goff St., Auburn
Frederick B. Lidstone, M.D., 117 Goff St., Auburn
Gerard L. Morin, M.D., 185 Webster St., Lewiston
Charles A. Hannigan, M.D., 85 Goff St., Auburn

Franklin County Medical Society

Delegates: Maynard B. Colley, M.D., 14 Main St., Farmington, Secretary
Stanley B. Covert, M.D., Kingfield

Alternate

Paul A. Brinkman, M.D., Farmington

Oxford County Medical Society

Delegates: Ake Akerberg, M.D., 2 E. Main St., South Paris, Secretary
Peter B. Aucoin, M.D., 151 Franklin St., Rumford (1 yr.)
James A. MacDougall, M.D., 303 Penobscot St., Rumford (2 yrs.)

Alternates

Walter G. Dixon, M.D., 16 Deering St., Norway (1 yr.)
Albert P. Royal, Jr., M.D., 82 Maine Ave., Rumford (2 yrs.)

THIRD DISTRICT

Knox County Medical Society

Delegates: Peter H. Holz, M.D., 51 Elm St., Camden, Secretary
Johan Brouwer, M.D., 5 Beech St., Rockland
Harry G. Tounge, Jr., M.D., 12 Union St., Camden

Alternates

William W. Ward, M.D., P. O. Box 804, Rockland
Wesley N. Wasgatt, M.D., 41 Talbot Ave., Rockland

Lincoln-Sagadahoc County Medical Society

Delegates: George W. Bostwick, M.D., Newcastle, Secretary
John F. Andrews, M.D., 20 West St., Boothbay Harbor
Ralph C. Powell, M.D., Damariscotta

Alternates

Charles E. Burden, M.D., 1 North St., Bath
Paul A. Fichtner, M.D., 781 High St., Bath

FOURTH DISTRICT

Kennebec County Medical Association

Delegates: Earle M. Davis, M.D., 2 School St., Waterville, Secretary
Kenneth W. Sewall, M.D., 2 School St., Waterville
William N. Runyon, M.D., 283 Water St., Augusta
Richard E. Barron, M.D., Main St., Monmouth
Valentine J. Moore, M.D., Thayer Hospital, Waterville
Paul A. Jones, Jr., M.D., 2 School St., Waterville

Alternates

Richard T. Chamberlin, M.D., 14 Gilman St., Waterville
Francis J. O'Connor, M.D., 4 Woodlawn St., Augusta
John M. H. Barnard, M.D., Doctors Park, 89 Hospital St., Augusta
Richard L. Chasse, M.D., 18 Park St., Waterville
Robert H. Dunn, M.D., Veterans Adm. Ctr., Togus

Somerset County Medical Society

Delegates: Marian L. Strickland, M.D., Easy St., Canaan, Secretary
George E. Sullivan, M.D., R.F.D. #1, Fairfield
Harland G. Turner, M.D., Box 38, Norridgewock

Alternates

Edgar J. Smith, M.D., 1 Park St., Fairfield
John P. Dow, M.D., 59 Main St., Pittsfield

Waldo County Medical Society

Delegates: Seth H. Read, M.D., 15 Church St., Belfast, Secretary
Norman E. Cobb, M.D., 132 Main St., Belfast

Alternate

George L. Temple, M.D., Fahey St., Belfast

FIFTH DISTRICT

Hancock County Medical Society

Delegates: Russell G. Williamson, M.D., Blue Hill Mem. Hospital, Blue Hill, Secretary
Russell M. Lane, M.D., P. O. Box "J", Blue Hill
Herbert T. Wilbur, Jr., M.D., 100 Main St., Southwest Harbor

Alternates

Philip L. Gray, M.D., Blue Hill
Llewellyn W. Cooper, M.D., 194 Main St., Bar Harbor

Washington County Medical Society

Delegates: Karl V. Larson, M.D., East Machias, Secretary
Hazen C. Mitchell, M.D., Calais

Alternate

James C. Bates, M.D., Eastport

SIXTH DISTRICT

Aroostook County Medical Society

Delegates: George J. Harrison, M.D., Market Square, Houlton, Secretary
Raymond G. Giberson, M.D., 156A Academy St., Presque Isle
R. Paul Johnson, M.D., Main St., Fort Kent
John B. Madigan, M.D., Houlton

Alternates

H. Douglas Collins, M.D., Caribou Clinic, Caribou
Melvin R. Aungst, M.D., Morneault Bldg., Fort Kent
George F. Higgins, M.D., 122 Academy St., Presque Isle

Penobscot County Medical Association

Delegates: Charles D. McEvoy, Jr., M.D., 186 State St., Bangor, Secretary
Carl E. Blaisdell, M.D., 47 Broadway, Bangor
Deane L. Hutchins, M.D., Health Dept., University of Maine, Orono
Arthur N. Lieberman, M.D., 180 Broadway, Bangor
Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor
George W. Wood, III, M.D., 156 No. Main St., Brewer

Alternates

George O. Chase, M.D., Eastern Maine General Hospital, Bangor
James D. Clement, Jr., M.D., 77 Essex St., Bangor
Clement S. Dwyer, M.D., 205 French St., Bangor
Irvin E. Hamlin, M.D., Main St., East Millinocket
William M. Shubert, M.D., 317 State St., Bangor

Piscataquis County Medical Society

Delegates: Odd S. Nielsen, M.D., 85 Pleasant St., Dexter, Secretary
Linus J. Stitham, M.D., 50 Main St., Dover-Foxcroft

Alternate

Charles H. Lightbody, M.D., No. Main St., Guilford

Special Exhibits

Albany Medical College

AMPAC and MEMPAC

Associated Hospital Service of Maine

Boston Medical Reports

Diverse Neuropathology in Mental Retardation – (Exhibit presented by Jose Galindo, M.D.)

Division of Cancer Control of the State of Maine Department of Health and Welfare

Division of Eye Care and Special Services of the State of Maine Department of Health and Welfare

Maine Cancer Society

Maine Chapter, American Academy of General Practice

Maine Dairy Council

Maine Heart Association, Inc.

Maine Hospital Association exhibit of Statewide Radio Communications for medical facilities

Maine Radiological Society

Maine Society of Clinical Hypnosis

Maine Thoracic Society – Emphysema – A New Definition

Maine Trauma Committee – Experience Using Neurovascular Pedicle Principle For Thumb Reconstruction

Mammography – (Exhibit presented by Charles W. Capron, M.D.)

The National Foundation – Birth Defects

New England Physicians Retirement Program

Vocational Rehabilitation

Technical Exhibits

Abbott Laboratories, North Chicago, Illinois
Representatives: Mr. A. J. Mack, Mr. A. Tancredi, Mr. W. A. Towne and Mr. P. F. Woodlock

Adco Surgical Supply Company, 8 Harlow St., Bangor, Maine
Representatives: Mr. Merrill R. Allen and Koger C. Anders

The Alkalol Company, Taunton, Massachusetts
Representative: Mr. E. W. LeClair

Americana Corporation, Northeast Division, 33 Ship Ave., Medford, Massachusetts
Representatives: Mr. Manny Misiph, Mr. Tony Bruno and Mr. Henry Sneider

Ayerst Laboratories, 245 Paterson Ave., Little Falls, New Jersey
Representative: Mr. Edward McMahon

Bicknell Photo Service Inc., 24 Free St., Portland, Maine
Representatives: Mr. G. Roger Baker, Mr. Fred Wormell and Mr. Ed Bicknell

Elmer N. Blackwell, Surgical Appliance Specialist, 565 Congress St., Room 207, Portland, Maine
Representatives: Mr. Elmer N. Blackwell and Mr. Oakley R. Sanborn

The Borden Company, 350 Madison Ave., New York, New York
Representative: Mr. J. R. Galvin

Brewer & Company Inc., 67 Union St., Worcester, Massachusetts
Representative: Mr. Walter Spaulding

Bristol Laboratories, 45 Newell Rd., Yarmouth, Maine
Representatives: Mr. William S. Read, Mr. Guy Hunter and Mr. Richard A. Green

Burroughs Wellcome & Co. (U.S.A.) Inc., 1 Scarsdale Rd., Tuckahoe, New York
Representatives: Mr. R. Parke, Mr. R. D. St. Laurent and Mr. E. J. Benoit

Ciba Pharmaceutical Company, 556 Morris Ave., Summit, New Jersey
Representatives: Mr. J. H. Angis and Mr. J. F. Sullivan

The Coca-Cola Company, P. O. Drawer 1734, Atlanta, Georgia

Encyclopaedia Britannica, 425 North Michigan Ave., Chicago, Illinois
Representatives: Mr. John Quinn and Mr. Franklin Strader

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Lederle Laboratories, Pearl River, New York
Representatives: Mr. Rocco Maffei and Mr. Robert G. Pelletier

Maine Surgical Supply Co., 233 Vaughan St., Portland, Maine
Representatives: Mr. John H. Lacy, Mr. Robert Axelsen, Mr. Lawrence Gardiner and Mr. Philip Dana, Jr.

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Representative: Mr. Joseph R. Ruest, Jr.

Mead Johnson Laboratories, Evansville, Indiana
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Representative: Mr. William P. Dunbar

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A. H. Robins Company, Inc., 1407 Cummings Dr., Richmond, Virginia
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Warner-Chilcott Laboratories, Morris Plains, New Jersey
Representative: Mr. Richard S. Marr

AMA NYC 1965

June 20-24, 1965

114th Annual Convention

DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page* 134

available, two children have been diagnosed as having PKU but neither were tested as newborns. Presumptive positive cases are referred to the Pineland Hospital and Training Center where the laboratory can provide more definitive laboratory studies for possible confirmation.

Following the discovery of maternal phenylketonuria,[1] mothers of infants giving tests of 4 mgm % or above, are requested to submit blood specimens for testing. In maternal PKU, the initial screening of the child is positive but follow-up tests are negative indicating a passive transfer of phenylalanine to the child. Exposure of the child to elevated phenylalanine levels during uterine life produces damage and retardation. Discovery of high phenylalanine levels in the mother, even though she is of normal intelligence, may allow for dietary adjustment during subsequent pregnancies and normal offspring. Investigations of the families of these women frequently are productive of additional cases of the maternal form of the disease as well as usual PKU cases.

Other inborn errors of metabolism have been described in the recent literature. Dr. Guthrie has developed screening tests for several of these conditions and two of them are now being evaluated in Massachusetts, southern California and Oregon: galactosemia and maple syrup urine disease (MSUD). Both of these conditions are known to be fatal but will cause retardation in the survivors. Histidinemia has been described in thirteen children all but one of whom had speech defects; only three were mentally retarded. Dr. Guthrie has suggested that new born infants of the future will be subjected to "biochemical heel printing." In this process, the infant's blood will be screened for all of the known inborn errors of metabolism known to cause preventable mental retardation and even death. Identifying these infants and placing them on diets appropriately adjusted to their respective defect will serve to reduce the more severely retarded portion of the population.

It is hoped the medical community of the state will cooperate closely with the Division of Maternal and Child Health and the Diagnostic Laboratory to insure the testing of all newborn infants in Maine.

REFERENCE

1. Mabry, C. C., Denniston, J. C. Nelson, T. L., and Son, C. D.: Maternal Phenylketonuria. New Eng. J. Med., 269: 1404-1408, 1963.

The Wonderful World of Drug Serendipity

Serendipity has always provided some of the greatest discoveries. Search for a seasickness remedy produced the antihistamines. Investigation of a hiccough remedy yielded a potent analgesic. Subsequently, the antihistamines were discovered to be antinauseants; then tranquilizers. Snake venoms, studied for the treatment of arthritis, were found to be anticoagulants and fungicides. The combined study of antibacterial drugs and cabbage feeding led to the antithyroid drugs. From these, it can be seen that the ultimate destiny of a new drug is not always predictable until its use is widespread. — A. Lee Lichtman, M.D., in **Experimental Medicine and Surgery,** 22:2-3, (Jun-Sep.) 1964.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Tissue Changes Are Reversible . . . If!

Animal experiments have shown that early pre-cancerous tissue changes will clear up completely once exposure to tobacco smoke condensate is ended. This research by Drs. Dale L. Tipton and T. Timothy Crocker of the University of California School of Medicine was reported by the American Cancer Society, which has helped support the work.

Other scientists have found at autopsy that cigarette smoking humans regularly develop several abnormalities of tissues lining the bronchial tubes, or bronchi. One of the most common of these is squamous metaplasia, the disorganized growth of the top layer of the bronchial lining. The scientists who discovered these changes – extremely common in smokers and relatively infrequent among non-smokers – regarded the changes as among the first in the development of lung cancer.

While the autopsies showed that people who had quit smoking had much less squamous metaplasia than those who continued to smoke, there was no actual proof that the diseased tissue had actually become healthy again. Drs. Tipton and Crocker set out to learn whether squamous metaplasia could revert to normal tissue.

The California scientists, using a bronchoscope, applied cigarette tars to the bronchi of anesthetized animals and then periodically examined the bronchial tissues. The tars were obtained from a cigarette smoking machine at Roswell Park Memorial Institute in Buffalo, N. Y. They had been condensed from the same kind of smoke inhaled by humans.

They found that in a startlingly short time – three days following tar application — drastic changes occurred in the bronchial lining. The cells which manufacture mucus which oils the tubes and engulfs foreign particles began to disappear over a wide area; and so did the cells which sweep out the tract with countless tiny brooms called cilia. In their place there arose the abnormal, pathologic cells forming the overgrown tissues of squamous metaplasia. There were smaller areas of other kinds of abnormal cells – transitional metaplasia and hyperplasia (simple overgrowth).

With no further exposure to cigarette tars, the different types of abnormal cells, which had covered extensive areas of the bronchus, slowly began to disappear; squamous metaplasia disappeared first. In their place, healthy mucus-producing and ciliated cells once again appeared.

Five weeks after tar exposure had ended, squamous metaplasia had receded so much that transitional metaplasia was more in evidence. At 14 weeks normal tissue had almost replaced the hyperplastic, or overgrown, tissues. At 18 weeks all tissues again were normal.

The experiments suggested that the cells most sensitive to the tars were not the mature-adult cells that line the surface of the bronchial passages but rather the basal cells. The order in which the tissue abnormalities disappeared — first in squamous tissue, then transitional, and finally, hyperplastic tissues — suggested that the whole disease process started in the basal cells.

The basal cells are the precursors of surface cells and are found below the surface cells. In the processes of normal regeneration, the basal cells differentiate into the mature surface cells as the older surface cells die and need replacement.

Under the influence of cigarette tars the basal cells became "juvenile delinquents" destined to become "adult criminals." They never did "reform," but as they died off they were replaced by a new generation of healthy basal cells which grew up into normal mature surface cells.

The scientists feel that something like this happens in the lungs of those who quit smoking – *if* they quit before malignant and irreversible cell changes take place.

Maine Heart Association Notes

Prophylactic Anticoagulant Therapy In The Orthopedic Patient

"It is well-established that patients subjected to immobilization are prone to thrombo-embolic phenomena. . . . The orthopedic patient should be particularly prone to thrombo-embolic complications as he is subjected to considerable soft tissue trauma, muscular trauma, and lengthy immobilization. . . .

". . . It would appear that the orthopedic patient facing a period of immobility should be anticoagulated to prevent thrombo-embolic complications. . . .

". . . In order to further delineate the benefits of prophylactic anticoagulants in orthopedic cases, the following study of 100 cases was undertaken.

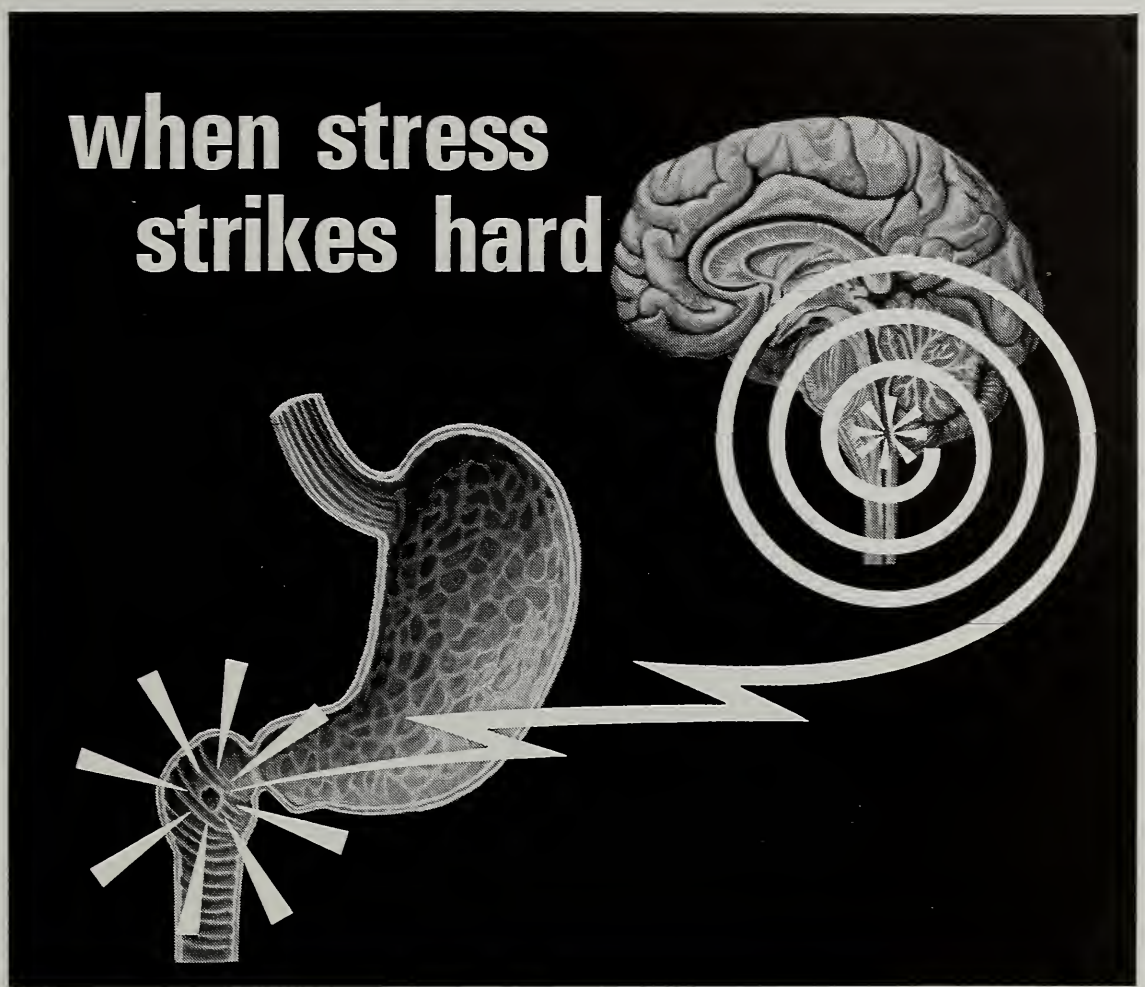
"One hundred consecutive orthopedic patients who suffered trauma of the pelvis and lower extremities were studied in regard to the value of routine prophylactic warfarin sodium anticoagulation in the prevention of thromboembolic phenomena. The variation between the 50 anticoagulated patients and the 50 control patients was statistically significant. (20% incidence of thromboembolic phenomena in nonanticoagulated cases and no thrombo-embolic episodes in the anticoagulated patients.) Prophylactic anticoagulation was found to be a safe procedure and is recommended for the adult patient with significant injuries about the hip and pelvis. Further studies of this problem in the younger age group are desirable."

Reference: Neu, Jr., L. T. et al. Annuals of Internal Medicine, Volume 62, pages 463-467, 1965.
Submitted by Jacob B. Dana, M.D., Gardiner, Maine.

A Guide To Anticoagulant Therapy

This booklet provides the physician with guiding principals and practical recommendations for the proper use of anticoagulant drugs. The material in this guide is for the physician who has decided to use anticoagulants and is designed to help him make clinical judgments. It does not consider the indications for therapy or the merits of different agents in the prophylaxis or treatment of specific diseases.

The booklet, which was prepared by the Committee on Professional Education of the American Heart Association is available, free of charge, on request, at the Maine Heart Association, Augusta, Maine.



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With Dartal (thiopropazate dihydrochloride) extrapyramidal and parasympatholytic symptoms have been reported and, rarely, leukopenia, erythematous skin reaction and allergic purpura. Do not administer to patients under the influence of alcohol, barbiturates or narcotics and use cautiously with sedatives, in epileptic or depressed patients or in those with liver damage. Reactions typical of phenothiazines may occur.

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Research in the Service of Medicine

Necrologies

William F. Mahaney, M.D.

1911-1965



William F. Mahaney, M.D., 54, of Saco, Maine, a Past President of the Maine Medical Association, died on May 4, 1965. He was born in Biddeford, Maine on March 31, 1911, son of William F. and Annie T. Mahaney. He graduated from Georgetown University in 1933 and received his medical degree from the University of Pennsylvania in 1937. He interned at the Kings County Memorial Hospital in New York from 1937 to 1939 and served a residency in Pathology at the New England Deaconess Hospital in Massachusetts from 1944 to 1945.

Dr. Mahaney was a member of the York County Medical Association and the Maine Medical Association. He was Councilor for the First District of the Maine Medical Association from 1951 to 1954, Council Chairman from 1953 to 1954 and President of the Association from 1954 to 1955. He was also a member of the American Medical Association, the Massachusetts Medical Society, a fellow of the American College of Surgeons, a Past President of the Maine Cancer Society, Chief of Surgery at Webber Hospital in Biddeford the past 15 years and a member of the Surgical Staff at Notre Dame Hospital in Biddeford.

Surviving are his widow, Mrs. Marion Evans Mahaney of Saco; three sons, 2nd Lt. William F., Jr., Ensign John Alan and Evan Evans; one sister, Miss Kathryn B. Mahaney; two brothers, John V. and Hilary F., all of Saco.

Wallace E. Webber, M.D.

1873-1964

Wallace E. Webber, M.D., 91, of Lewiston, Maine died on September 15, 1964. He was born in Topsham, Maine on August 14, 1873, son of Arista and Louella P. Wedgwood Webber.

Dr. Webber graduated from Edward Little High School in 1891 and received his medical degree from Bowdoin Medical School in 1895. He entered private practice in Lewiston in 1895 and was admitted to the staff of the Central Maine General Hospital where he served as Adjunct Surgeon from 1896 to 1903. He was Attending Surgeon on the active staff from 1903 to 1926 and Consulting Surgeon from 1926 until his death. Dr. Webber became the first physician in the Lewiston area to limit his practice to surgery.

Dr. Webber was an Honorary member of the Androscoggin County Medical Association and the Maine Medical Association, having received a 50-year pin in 1945, a 55-year pin in 1950, a 60-year pin in 1955 and a 65-year pin in 1960. He

was also a member of the American Medical Association, a charter member of the Lewiston Rotary Club and in 1952 was honored by the Lewiston Chamber of Commerce for fifty years of service to the community.

Dr. Webber is survived by two daughters, Miss Dorothy P. Webber of Lewiston, Maine and Mrs. Lydia R. Rogers of Natick, Massachusetts.

Harris C. Barrows, M.D.

1881-1965

Harris C. Barrows, M.D., 84, of Augusta, Maine died on May 12, 1965. He was born in Ludlow, Vermont on February 1, 1881, son of William E. and Annie Clark Barrows.

Dr. Barrows graduated from Cony High School in 1899 and received his medical degree from Bowdoin Medical School in 1906. He did postgraduate work at the Central Maine General Hospital in Lewiston from 1906 to 1907 and the Gardner State Hospital in Massachusetts from 1908 to 1910. He practiced medicine at Boothbay Harbor for fifty years before retiring.

Dr. Barrows was an Honorary member of the Lincoln-Sagadahoc County Medical Society and the Maine Medical Association, having received a 50-year pin in 1956 and a 55-year pin in 1961. He was a member of the American Medical Association, the Seaside Lodge, F and AM, Boothbay Harbor, the Gen. Knox Chapter Scottish Rite bodies of Rockland, the Maine Consistory of Portland and was a 32nd Degree Mason. He was also a life member of Knights of Pythias and the Charles E. Sherman Post, AL of Boothbay Harbor.

He is survived by a sister, Mrs. Alida Barrows Reynolds of Augusta and several nieces and nephews.

In Memoriam

Androscoggin County	
Wallace E. Webber, M.D.	Lewiston
Cumberland County	
Theodore C. Bramhall, M.D.	Sarasota, Florida
Kennebec County	
Roland L. McKay, M.D.	Augusta
Charles E. G. Shannon, M.D.	Waterville
Knox County	
Armand Albert, M.D.	Owl's Head
Merrill J. King, Sr., M.D.	Portland
Lincoln-Sagadahoc County	
Harris C. Barrows, M.D.	Augusta
Penobscot County	
Herbert C. Scribner, M.D.	Bangor
Albert C. Todd, M.D.	Brewer
York County	
William F. Mahaney, M.D.	Saco
James W. Sever, M.D.	Cambridge, Massachusetts

County Society Notes

SOMERSET

The Somerset County Medical Society met at the Oak Pond Restaurant in Canaan, Maine on December 15, 1964.

The meeting was called to order by H. Carl Amrein, M.D., Vice-President.

Attendance at the annual convention of the Maine Medical Association at Rockland was discussed. The opinion was expressed that it was too bad to have such poor attendance at the various meetings when an effort is put forth to procure top-notch speakers.

The business meeting was adjourned and the time given to William B. Grow, M.D., Superintendent of the Central Maine Sanitorium. Dr. Grow presented several case histories of patients who have been committed to the sanitorium with active tuberculosis during the past few years. Dr. Grow's emphasis was that this disease is not a thing of the past and that any physician must be constantly on the alert for such cases. In one instance because of spread from one case, six active cases were hospitalized and treated at the sanitorium. At least eighty contacts were investigated and twenty given prophylactic therapy outside of the hospital. This happened in 1961. Charles D. McEvoy, Jr., M.D. of Bangor was present as a guest and answered questions and expressed his views.

An additional feature following a buffet dinner was the showing of colored slides of Italy, Scandinavian countries and some of the Holy Land by Dr. A. E. Rodden, D.D.S. of Skowhegan.

A meeting of the Somerset County Medical Society was held on February 16, 1965 at the Village Candlelight in Skowhegan, Maine.

H. Carl Amrein, M.D., Vice-President, called the meeting to order.

The meeting consisted mainly of business matters brought to the attention of the society by George E. Sullivan, M.D. He spoke about the matter of re-registration of Maine physicians and also of the reciprocity between states with regard to licensing of physicians.

The A.M.A.'s Eldercare plan was described at some length and compared with the Medicare plan.

W. Edward Jordan, Jr., M.D., President, read a letter from the Semper Fidelis Club which requested approval from the medical society for their sponsorship of an oral polio vaccine clinic in Skowhegan. A motion was made and carried for more information to be obtained with regard to this program.

The meeting was adjourned and members met together informally at dinner.

The Somerset County Medical Society met at Whittemore's Restaurant in Skowhegan, Maine on April 27, 1965.

The meeting was called to order by H. Carl Amrein, M.D., Vice-President.

During the business meeting George E. Sullivan, M.D. brought several matters to the attention of the society from the Interim Meeting of the Maine Medical Association House of Delegates which was held April 11. Among the matters brought forth was the budget for the fiscal year 1966, availability of Malpractice Insurance through the Aetna Company, and report of the postgraduate television program. A motion was made and approved that the delegates be instructed to vote for approval of the budget as submitted.

Edgar J. Smith, M.D. spoke about insurance with regard to Blue Shield fees for psychiatrists and services in other areas, the enrollment of members – open or group, and the point system in arriving at fees. A motion was made and carried

COUNTY SOCIETIES

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President, Henry C. Thacher, M.D., Auburn
Secretary, Donald L. Anderson, M.D., Lewiston

Aroostook

President, R. Paul Johnson, M.D., Fort Kent
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Secretary, Russell G. Williamson, M.D., Blue Hill

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Secretary, Odd S. Nielsen, M.D., Dexter

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Secretary, Marian L. Strickland, M.D., Canaan

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Secretary, Karl V. Larson, M.D., East Machias

York

President, Alvin A. Hoffman, M.D., York
Secretary, Charles W. Kinghorn, M.D., Kittery

that the society designate our insurance representative, Dr. Smith, to vote for the point system.

A letter from the secretary of the Semper Fidelis Club of Skowhegan detailing a proposed clinic to offer oral polio vaccine to pre-school and school children to age 18 was read. A motion was made and carried that the society inform the club that although the majority approved of such a program, that in this case the implementation of such a clinic was in the jurisdiction of the Public Health Department of Skowhegan.

The special speaker for the evening was Mrs. Dorothy Clarke Wilson of Orono, Maine. Mrs. Wilson has written several books on medical personages who have been foremost in helping to give to the people of India better medical care. Mrs. Wilson showed colored slides of the Christian Medical School in Vellore, India. This school is supported by 50 different denominations and was started by Dr. Ida Scudder a few years after the turn of the century. Mrs. Wilson spent several months in Vellore before Dr. Ida passed away (1960) gathering material for her book, *DR. IDA.* She also has written about a young woman physician, who following an unfortunate accident, became a paraplegic. *TAKE MY HANDS* is the story of Dr. Mary Verghese who came to this country several years ago to train in the field of rehabilitation and who now heads such a department at the Christian Medical School. The work of rehabilitation of lepers was shown in slides under the direction of Dr. Paul Brand who is a leader in this field, especially in surgical rehabilitation. A book about his work by Mrs. Wilson will shortly go to press. The program was most interesting and very well presented. Following Mrs. Wilson's lecture the members and their wives enjoyed dinner together, with the Reverend and Mrs. Wilson as guests.

MARIAN L. STRICKLAND, M.D.
Secretary

CUMBERLAND

The Cumberland County Medical Society met at the Pineland Hospital and Training Center in Pownal, Maine on April 15, 1965 with fifty-seven members and several guests attending.

Following a social hour and dinner, the President, Alvin A. Morrison, M.D. presented Peter W. Bowman, M.D., Hospital Superintendent, who introduced the speakers and their topics:

Jose Galindo, M.D., *The Clinical Laboratory and the Retardation Syndrome;* Edward Blumberg, M.D., *Rare Clinical Syndromes in Patients with Mental Retardation;* Aldo F. Llorente, M.D., *Childhood Autism and the Retardation Syndrome. Shared and Differential Signs* and Richard Holmes, M.A., *Audiology and Speech Evaluation as a Segment of Differential Diagnosis in Mental Retardation.*

David K. Lovely, M.D. summarized the recent meeting of the House of Delegates of the Maine Medical Association presenting background information to lead to instruction of delegates at the May meeting.

STANLEY B. SYLVESTER, M.D.
Secretary

ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at the Montello Manor in Lewiston, Maine on April 15, 1965.

The meeting was called to order by the Vice-President, Harvey J. Proulx, M.D. in the absence of the President, Henry C. Thacher, M.D.

Thomas A. Martin, M.D., President of the Maine Medical Association, was introduced.

Dean H. Fisher, M.D., Commissioner of State Bureau of Health and Welfare, speaker of the evening, spoke relative to Medicare. A question and answer period followed.

D. F. Russell, M.D. is to receive his 60-year pin from the Maine Medical Association at the June meeting.

Edward L. Reeves, M.D. reported on the Interim Meeting of the Maine Medical Association House of Delegates.

DONALD L. ANDERSON, M.D.
Secretary

Deceased

Roland L. McKay, M.D., P. O. Box 265, Augusta, Maine, May 24, 1965

News, Notes and Announcements

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine
March 9-11, 1965

THROUGH EXAMINATION

Otto Appenzeller, M.D., 42 Rangeley Rd., Winchester, Mass.

Maurice B. Chammas, M.D., Rumford Community Hospital, Rumford, Me.

Ezatollah Foroughi, M.D., 180 Maple St., Needham, Mass.

Michael M. Geldner, M.D., 6 Lenox St., Edison, N. J.

Daryush Haghighi, M.D., Southwest Community Hospital, Berea, Ohio

Roger E. Hakim, M.D., Akron City Hospital, Akron, Ohio

Oscar Hantz, M.D., 1826 Metzerrot Rd., Adelphi, Md.

Gholam H. Khoshnevisan, M.D., 1116 Frederick Blvd., Akron, Ohio

Nauttam J. Kothari, M.D., 120 East 36th St., New York, N. Y.

Michael I. Leckie, M.D., 1377 Washington Ave., Portland, Me.

Harilaos T. Sakellarides, M.D., 481 Boylston St., Brookline, Mass.

Manuel O. San Jose, M.D., Bellevue Hospital Center, New York, N. Y.

Sally Sut-Yee Sun, M.D., V. A. Hospital, Fort Howard, Md.

Antonio Valero-Ramo, M.D., New England Medical Center, Boston, Mass.

Wilhelmus A. Van Haaren, M.D., St. Luke's Hospital, New Bedford, Mass.

Hasan F. Yaman, M.D., St. Sauveur Hospital, Val D'Or, U.Q., Can.

Davor Zuberko, M.D., 433 West 46 St., New York, N. Y.

THROUGH RECIPROCITY

Robert E. McAfee, M.D., 150 Ludlow St., Portland, Me.

Sunny J. Bullington, M.D., R.D. #4, Danville, Pa.

Milton H. Clifford, M.D., V. A. Hospital, Rutland Hts., Mass.

John H. Derry, M.D., 65 E. 93 St., New York, N. Y.

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The Journal
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Factors in the Prevention of Abdominal Wound Dehiscence

William M. Stahl, M.D.*

Since the first surgical celiotomy, undesired rupture of the postoperative abdominal incision has remained a catastrophic occurrence. Although varied studies have been done to delineate the physiologic and biochemical factors involved in the healing of abdominal wounds, and although many clinical advances in patient support have been achieved, dehiscence of the abdominal wound occurs all too frequently and carries significant mortality. Reported series indicate that this complication occurs in from .51% to 5.8% of all major abdominal incisions, and that one may expect a mortality of from 11% to 30% due to such occurrence.

Since the early work of Howes, Sooey and Harvey in 1929,[11] basic factors of importance in wound healing have been studied. The phases of cellular infiltration, fibroplasia, and collagen deposition, and the chemical anatomy involved in the production of muco-polysaccarides have become known. Investigators have demonstrated the importance of adequate protein nutrition, especially methionine, in the formation of capillaries, proliferation of fibroblasts and production of ground substance and collagen.[25,14] The necessity for adequate stores of ascorbic acid has been shown for adequate collagen synthesis.[6] The adverse effect of untreated metabolic disturbances such as diabetes has been documented,[20] as has the adverse effect of excess adrenal cortical secretion.[2,1]

The tremendously important factors of gentleness of surgical technique, care in handling of wound edges, use of fine ligature material, removal of avascular and traumatized tissue and careful closure without tension have been stressed by Dunphy and Jackson[5] and amply supported by experimental and clinical studies. Some interference in healing has been noted following exposure to ionizing radiation.[12,10]

Studies of healing wounds directed toward specific types of suture material have confirmed the superiority of fine, non-absorbable sutures over catgut.[19,15,5] Absorbable sutures have been found to cause granulomatous reactions in some wounds.[9] Apparently this is more commonly seen in sutures sterilized by electron beam as compared to heat sterilized material. The difficulties of healing infected wounds containing non-absorbable sutures, however, is a problem. Although steel wire has been significantly better in this regard there have been some reactions.[21] Nelson and Dennis explored the technical factors in the production of wound strength[18] and concluded that the suture material constitutes all of the strength of the healing wound during the lag period and is important up to 14 days. They, too, found stainless steel wire closed wounds to have an increased strength and less reaction than those closed by other methods.

Specific etiologic factors, in addition to general wound physiology, have been sought since Freeman in 1927 proposed that these dehiscences were primarily caused by peritoneal defects.[8] This thesis has remained popular although never well proved. Drye,[4] in 1948 showed that ambulation and arising from bed were minor factors in increasing intraperitoneal pressure compared to straining at stool, coughing or vomiting; these latter produced five to tenfold increase over the supine abdominal pressure. Forsell[7] studied postoperative fascial separation in upper

*Associate Professor of Surgery, The University of Vermont College of Medicine, Burlington, Vermont

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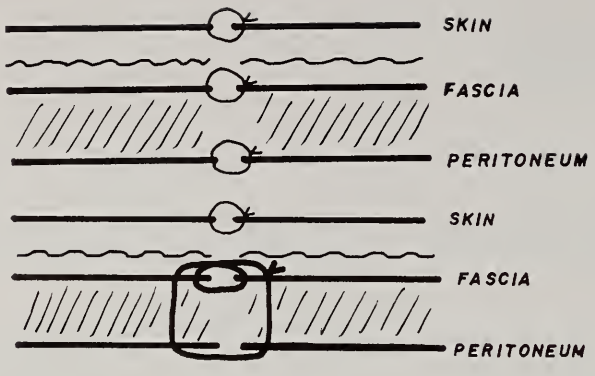


FIG. I

Diagramatic Representation of Wound Closure Techniques

Upper figure shows interrupted closure of all layers. Lower figure shows buried wire closure far and near type of fascia and peritoneum. Closure of subcutaneous tissue is usually done with fine interrupted sutures but is omitted from the figure for clarity.

abdominal incisions comparing vertical midline incisions with oblique incisions and found less separation in the oblique wound.

Most recent reports of abdominal wound dehiscence agree on these major points of wound healing in general, and healing of abdominal wounds in particular.[26,24,16,13,17,22] These authors found strong association with postoperative cough, vomiting and abdominal distention, with infection playing a lesser role. Most of the abdominal disruptions occurred in the older age group with a three to five times higher incidence in patients older than 45 to 50 years. A high incidence in cases of carcinoma was noted. An increased incidence was present in protein depleted patients, and in the early part of the year, possibly associated with poor winter dietary habits.

The majority of the abdominal wound disruptions occurred between the 6th and the 9th postoperative days, but 20% to 25% occurred at the end of the 2nd week and throughout the 3rd week of convalescence. Although Wolff reported a 3.2% incidence of dehiscence in cases where retention sutures were used, other authors[17,22] have felt these to be valuable and both Wolff and Tweedie suggest the use of buried wire sutures. Spencer et al has called attention to this type of closure in a recent report of 293 cases with only one dehiscence.[23]

Reduction of the incidence of postoperative wound dehiscence to the minimum would then demand optimum preparation of the patient, especially with regard to protein and vitamin nutrition, maximal improvement in any preexisting pulmonary disease, adequate control of diabetes or other metabolic disorders, avoidance of midline incisions, the use of transverse or oblique incisions where possible and convenient, atraumatic wound care, proper closure, and prevention, if possible, of postoperative vomiting, distension and cough.

Table 1

Age and Sex Distribution

	Female	Male	Total
0-20	23	29	52
20-30	33	13	46
30-40	62	20	82
40-50	78	32	110
50-60	69	33	102
60-70	87	34	121
70-80	45	20	65
over 80	10	12	22
Total	407	193	600

Table 2

Types of Operations

	Female	Male	Total
Appendix	32	42	74
Hernia	26	13	39
Biliary Tract	143	40	183
Stomach	21	32	53
Intestine	32	30	62
Colon	50	16	66
Pelvic	90	—	90
Vascular	1	11	12
Other	12	9	21
Total	407	193	600

Table 3

Incisions

	Female	Male	Total
Vertical			
Rectus	180	93	273
Midline	95*	11	106
Transverse	119	81	200
Other	13	8	21
	407	193	600

(* 90 pelvic operations)

Table 4

Methods of Closure

	Female	Male	Total
Silk interrupted	216	98	314
Catgut & silk	54	27	81
Wire	137	68	205
	407	193	600

Exceptional diligence with regard to these factors should be exercised in the closure of wounds in elderly patients, patients with unreplacable protein deficits, patients who have been on cortico-steroid therapy or are undergoing unusually severe degrees of trauma or stress, patients who require long or complicated incisions and lengthy operative procedures, patients where infection may supervene and where drains or "ostomies" are needed (these should be put through separate wounds where possible) and patients where distension, acites, effusion or cough may be unavoidably present.

With these dicta in mind and with special attention

to (1), the factor of peritoneal defect in initiating dehiscence, (2), the good results documented in the use of wire suture, both as regards tensile strength of wounds and the behavior of wire in the presence of infection, and (3), the fact that dehiscence may occur well into the third postoperative week; the following routine of wound closure has been used in a personal series of 600 consecutive abdominal wound closures. All of these incisions are major laporotomy incisions, with no muscle splitting incisions included. The hernias noted are abdominal incisional hernias, no inguinal hernias being tabulated. Many of these incisions were long and complicated as for aortic or multiple organ resections. They are completely consecutive and unselected. The preferred closure in this series was that of interrupted 2-0 silk closure for peritoneum and fascia, and 4-0 silk closure for subcutaneous and skin. In a smaller percentage of cases early in the series interrupted catgut was used on the peritoneum and interrupted silk in the fascia. In cases of increased potential for abdominal wound dehiscence, buried far and near #28 stainless steel wire was used with 4-0 silk in subcutaneous and skin. These are a similar group to those described by Spencer. Two cases have required the removal of wire sutures due to tender palpable wire knots in thin abdominal walls. Two cases have required reopening of the wound and removal of infected granulomas surrounding silk suture material. There have been no dehiscences in this series.

Summary

A consecutive series of 600 patients with major abdominal incisions has been reviewed. There were no dehiscences in this series. The importance of general preparation of the patient, careful operative technique and wound closure, and close postoperative supervision have been stressed. Specific points of technique felt to be important are those of the use of interrupted nonabsorbable sutures for peritoneal and fascial closure in the good risk patient, and the use of buried far and near stainless steel wire sutures in the poor risk patient. It is felt that this technique has contributed to the absence of dehiscence in this series.

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Common Duct Reconstruction Outline For Treatment

B. Bernard Kaye, M.D.*

Trauma to the common duct may cause partial or complete severance with stricture formation or loss of continuity of the duct. Strictures of the common duct may also result from congenital anomalies or inflammatory lesions involving the walls of the common or hepatic ducts.

Mechanical obstruction of the ducts results in hepatobiliary dysfunction, chronic bile stasis and impairment of metabolic function. Cholangitis, periductal abscess and cicatricial tissue distorts and blocks the anatomical structures, giving rise to biliary cirrhosis with or without portal hypertension. The clinical picture is one of pain, recurrent chills and fever with jaundice. Restoration of the anatomy and physiology will eliminate this clinical syndrome.

Operative Cholangiography will localize the point of obstruction and the status of the proximal and distal duct segments.[1]

I. Etiology

A. Operative trauma[2] during Cholecystectomy is the most common cause of benign strictures of the extrahepatic bile ducts. Walters[3] reports stricture due to surgical injury in 264 out of 265 cases.

1. Tenting of the common duct with excision of a segment of the duct.
2. Attempts to control hemorrhage from the cystic or right hepatic artery.[4]
3. Application of clamps or sutures, crushing the common duct.
4. Failure to recognize an anomalous duct or vessel. The extrahepatic biliary system does not conform to a standard anatomical pattern, about 22% have some variations in size, shape or position of the ductal and vascular components.[5]
   a. A short cystic artery arising from a right hepatic artery in proximity to the cystic duct.
   b. A double cystic artery.
   c. A cystic duct emptying into a right hepatic duct or an accessory duct.
   d. Absence of the cystic duct.

B. Operative trauma during resection of Duodenal Ulcer, Peri-Vaterian Duodenal Diverticulum, Porta-Caval Anastomoses.

C. Liver trauma with collection of blood and bile in the region of the duct may cause fibrosis of the duct.

D. Trauma to the extrahepatic ducts with rupture or severance. Rupture of the hepatic, cystic or common ducts may cause fibrosis and obstruction due to bile being deposited about the common duct.

E. Congenital strictures in the newborn.

F. Inflammatory lesions.[6]

1. Inflammatory lesions and calculi of the duct may cause fibrosis of the tissues in and about the duct with resultant segmental stenosis of the duct.
2. Pancreatitis may cause inflammatory narrowing of the common duct.
3. Duodenal ulcer may cause inflammatory narrowing of the common duct.

II. Prevention

Biliary tract surgery requires adequate light, exposure, retraction, visualization and identification of all biliary tract structures prior to ligation.

Exposure of the cystic artery and cystic duct at its junction with the common duct. Ligation of the cystic artery prior to cystic duct ligation.

Recognition of anomalous anatomy.

Avoidance of stripping of the common duct of its enveloping sheath to prevent compromise of the blood supply to the duct with subsequent necrosis.[7]

III. Preoperative Preparation

Proper evaluation of the physio-pathology is essential, prior to undertaking a long and difficult technical procedure.[8] Adequate preoperative preparation necessitates investigation of hepatic function. Prothrombin time, serum bilirubin, protein, albumin-globulin ratio, alkaline phosphatase, cholesterol, cephalin-flocculation and thymol turbidity test will determine the extent of liver damage and the degree of surgical tolerance.

Preoperative use of Vitamin K, Fibrinogen, Serum Albumin and whole blood are utilized to correct hepatic deficiencies. Protracted jaundice with hepato-cellular involvement contradicates a prolonged operative procedure.

Decompression of the obstructed biliary system is essential. Preliminary choledochostomy allows decompression of the liver and hastens liver recovery. Not all duct operations can be corrected at a single operation, therefore a two stage procedure may be advisable. Creation of a free external biliary fistula permits subsequent exploration of the common duct. Preliminary Spleno-Renal shunt may be necessary to lessen the portal pressure, thereby minimizing blood loss during the definitive repair.

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IV. Surgical Repair or Reconstruction

There is no unanimity of opinion regarding the type of repair that is best utilized.

Lahey,[9] Donaldson, Allen and Bartlett[10] recommend primary end-to-end repair. Walters[3] favors ductal-duodenal anastomoses. Cole[11] favors ductal-jejunal anastomoses. Longmire[12,13] advocates intrahepatic cholangiojejunostomy in those cases where the upper segment is intrahepatic.

Despite the many operative technics designed to reconstruct the biliary passages, approximately 20 to 40% of repairs fail to produce a lasting satisfactory result.[14] Walters[3] reported 70 to 75% of cases had continued relief of biliary obstruction without recurrence of intrahepatic infection unless contraction of the anastomoses occurred.

The pathology found at the time of operative exploration will determine the type of repair or reconstruction to be utilized. The type of reconstruction depends upon the anatomo-pathological status of the intrahepatic bile ducts and the common duct. The operator must be familiar with all possible methods of repair or implantation of the bile duct into the intestinal tract.

The prognosis of common duct reconstruction depends upon the ability to dissect out the proximal duct, and the length of the common duct available for anastomoses. Fibrous and scarred tissues are resected to permit normal mucosa-to-mucosa approximation.

A. Surgical Approach

The surgical approach is through a right rectus[15] or subcostal incision[16] away from the previous pathology, to expose the hilum of the liver. The peritoneum is opened at the inferior portion of the incision. The hepatic flexure is usually adherent to the undersurface of the liver and is mobilized downward to the left after severance of the right Phrenico-colic ligament.

The second and third portions of the duodenum are elevated upward until the spermatic or ovarian vein, inferior vena-cava and aorta can be visualized from right to left. The duodenum is mobilized, rotated and displaced to the left, while the liver is retracted to the right, exposing the ventral surface of the liver.

The hilum is approached from right to left, and the bile ducts are traced as they enter the Porta-Hepatis. A drop of bile at the hilum may pinpoint the duct above the stricture. Any bulging tissue at the hilum lateral to the hepatic artery is aspirated. The difficult case may require dissection and identification of the hepatic artery.

The common duct is explored proximal to the stricture, the dissection from the liver parenchyma is kept close to the ducts. The upper portion of the duct cannot be located in about seven percent of cases.[3]

The dilated proximal duct is identified and 5 cc. of contrast medium is injected. The resulting cholangiogram outlines the stricture and reveals the length of the proximal duct segment available for repair. Multiple operative procedures may result in loss of substance of the hepatic ducts, with a short cuff remaining, necessitating dissection of the intrahepatic ducts out of the substance of the liver.

Extra length may be obtained by conversion of the right and left hepatic ducts into a common hepatic duct by suturing the ducts, crushing the spur and insertion of a split T tube into these proximal ducts.

The lower portion of the common duct is exposed and identified in the supra-duodenal area. The lower end of the common duct is located superior and medial to the lymph gland which lies on the posterior inferior aspect of the gastro-hepatic ligament, and serves as an accurate guide to the lower end of the common duct.[15]

The lower end of the common duct can be located by mobilizing the duodenum and head of the pancreas, making it accessible for repair without tension. The distal common duct can be mobilized by splitting the head of the pancreas.

A second operative cholangiogram is obtained after injection of 5 cc. of contrast medium. If the common duct is patent, and if the ampullary mechanism is functioning normally, continuity can be established.[9]

Stricture of the duct at the ampulla of vater is approached through the duodenum with division of the stricture under direct vision, followed by a transverse closure of the duodenum.

B. Duct-To-Duct Anastomoses (Choledocho-Choledochostomy)

Accurate mucosa-to-mucosa approximation without tension and avoidance of stasis are essential in the repair of the duct.

End-to-end anastomoses preserves choledochal sphincteric function and permits the Sphincter of Oddi to remain in functional continuity in the extrahepatic biliary system. End-to-end anastomoses may result in marked thickening and dilitation of the proximal segment, with thickening at the site of anastomoses, and subsequent stricture formation. There is some degree of inflammatory reaction in duct-to-duct anastomoses.

Some surgeons feel that primary end-to-end anastomoses performed at the time of injury has little chance of success, and therefore prefer anastomoses of the common duct to the jejunum as the method of choice. High duct injury is best treated by hepatico-jejunostomy.

Following identification of the common duct, scar tissue is excised, stones are ruled out in the intrahepatic ducts or ampulla, and a mucosa-to-mucosa repair is performed over a T tube, using one row of interrupted mattress catgut sutures. The T tube is brought out through an opening in the duct below the site of the anastomoses, thereby protecting the suture line against the resistance of the Sphincter of Oddi.

C. Biliary-Entero Anastomoses

Bile is channelled into the intestinal tract by anastomoses of the proximal portion of the common duct to either the duodenum or jejunum. This procedure is

utilized when a considerable portion of the common duct has been narrowed or destroyed by trauma or infection, and the lower end of the duct cannot be located.

Biliary intestinal anastomoses may result in some degree of cholangitis due to reflux of the intestinal content into the biliary tract.

Stricture of the common duct in the presence of the gall bladder requires cholecystectomy at the time of Biliary-Entero anastomoses to avoid cholecystitis or cholelithiases.

1. Choledochodudenostomy: Hepaticoduodenostomy

This procedure is applicable for strictures of the common duct, due to the close proximity of the duodenum to the common duct. The stump of the proximal duct is isolated at the hilum, and the duodenum is mobilized to approximate the duct. Accurate anastomoses of the mucosa of the duct to the mucosa of the duodenum is accomplished by a single layer of interrupted 2-0 chromic catgut sutures, or an outer layer of interrupted silk sutures and an inner layer of interrupted catgut sutures.

Anchoring of the duodenum to the liver by three or four interrupted sutures relieves tension on the suture line.

2. Choledochojejunostomy: Hepaticojejunostomy

Accurate apposition of the mucosa of the bile duct to the mucosa of the jejunum, and an adequate size stoma without tension are essential; otherwise the mucosa of the bile duct pulls away from the jejunal mucosa, leaving a gap which will heal with granulation tissue, then fibrous tissue, and the contracting scar will leave a stenosed anastomoses.[17] The anastomoses requires prolonged splinting to prevent contraction of scar tissue.

a. Cole[11] advocates the use of a defunctionalized loop of jejunum, utilizing twelve inches of jejunum between the duct and the intestinal anastomoses, thereby preventing regurgitation of food into the hepatic duct. Anastomoses of the proximal end of the common duct to the open end of the jejunum is performed, using the latter as an artificial common duct. A rubber catheter is brought out through an opening in the jejunum. No attempt is made to locate the distal duct.
When the intrahepatic duct cannot be located, a cone shaped piece of liver is cut over the duct, and the tip of the Roux-Y arm of jejunum is inserted into the opening. The serosa and muscularis are trimmed away from the tip of the jejunum to permit the mucosa tip into the opening.[18]
b. Cattell[15] utilizes anastomoses of the proximal end of the common duct to an antecolic loop of jejunum, using one layer of interrupted mattress catgut sutures over a prosthesis. A distal jejunojejunostomy is performed six to eight inches below the biliary-entero anastomoses.
c. Kirtley[19] has suggested anastomoses of the open end of the distal limb of jejunum to the hilum of the liver around but not to the proximal duct. The proximal duct is fishmouthed, anterior and posterior flaps are sutured to the thickened Glisson's capsule. Continuity of the intestinal tract is completed by an end-to-side Roux-Y jejunostomy. This procedure avoids a circular mucosal anastomoses between the bile duct and intestine. The largest possible opening is secured and maintained by bisecting the proximal duct.
d. Smith[17] utilizes intrajejunal anastomoses. Precise apposition of mucosa to mucosa at all points around the circumference of the anastomoses is achieved with complete inversion of the anastomoses into the jejunum to prevent early leakage or late stenosis. Seven to ten mm. of bile duct is mobilized outside the liver in order to construct the intrajejunal anastomoses. The open end of a Roux loop is lifted into the porta hepatis. The jejunum is opened five to seven cms. from the open end. One layer mattress suture anastomoses is performed through the open end of the Roux loop. The open end of the Roux loop is closed and the loop is suspended from the peritoneum or capsule of the liver.
e. Smith[20] has suggested transhepatic intubation, the tube emerging from the anterior surface of the liver. The tube allows diversion of bile from the anastomoses by suction in the postoperative period and daily washing of the ducts. The scarred common hepatic duct is dilated and the intrahepatic ducts are cleared of small stones. An angled forceps is introduced into the common hepatic duct and then into the left hepatic duct. The forceps can be introduced into one or two main subdivisions running close to the anterior surface of the liver and protrude from the liver. A Latex tube is brought down through the duct system and out of the open common hepatic duct. A Roux loop is brought up to the porta hepatis. The end of the loop is closed, and an opening is made in the anterior surface, the same size as the open end of the common hepatic duct. Hepatico-Jejunal anastomoses is performed. When half the anastomoses is constructed, the tube is inserted into the jejunal loop and the anastomoses is completed.
Occasionally ductal injury is so high in the portahepatis that there is no remaining common duct, and the right and left hepatic ducts have to be anastomosed to the jejunum. Bilateral transhepatic tubes are employed. The tube coming out of the anterior surface of the liver is brough out through a separate stab incision in the anterior abdominal wall. The abdomen is closed with drainage of the subhepatic space. The transhepatic tube is connected to a continuous electric pump and the subhepatic drain is connected to a vacuum pump. Postoperative suction prevents

leakage and pooling of infected bile around the anastomoses.

f. Longmire[12] utilizes intrahepatic cholangiojejunostomy for extensive fibrosis of the extrahepatic biliary tree in which the usual procedures for reestablishing biliary-entero continuity has failed. Partial resection of the left lobe of the liver is performed with anastomoses of one of the major intrahepatic biliary ducts to the jejunum. The rational lies in the concept that the ductile systems in the right and left hepatic lobes are in direct continuity and that by tapping the major biliary channel in one lobe, one may anticipate adequate biliary drainage from both major lobes of the liver.

Cattell[15] believes that the anastomoses to the left duct does not afford relief of obstruction of the great mass of liver tissues found in the right lobe.

Longmire[12] utilizes a left subcostal incision, division of the triangular ligament, and wedge shaped segment resection of liver. The suspected biliary ducts are catheterized and a intrahepatic cholangiogram outlines the intrahepatic biliary system to determine whether or not the right and left hepatic lobes will drain through the exposed biliary duct.

The distal limb of a Roux-Y segment is closed in two layers and the lateral border is anastomosed with the intrahepatic duct, using interrupted 4-0 chromic catgut sutures. The intraluminal catheter is brought out through the jejunum about four to six inches below the site of anastomoses, and then through a stab wound in the anterior abdominal wall.

Gastro-intestinal continuity is reestablished below the transverse mesocolon by an end-to-side jejunojejunostomy. Two Penrose drains near the anatomoses are brought out through a separate stab wound. The catheter is removed in ten to twenty-one days.

V. Splints

There are various types of splints utilized in restoration of common duct function. The tube should have a diameter about one-half the size of the duct it drains.

1. Rubber catheters which are passed in the intestinal lumen when the silk suture is cut.

2. Mayo-Sullivan, Y and Vitallium tubes are not passed and eventually become occluded with bile pigments and debris, and therefore when used should be planned as a two stage procedure. The prosthesis covered by heavy encrustation of bile salts contributes to the recurrence of the fibrosis. Cattell[15] has discontinued the use of Vitallium tubes.

3. T tubes are usually used in Choledocholedochostomy and Choledo-Enterostomy. Silicone T tubes (21) are inert, do not deteriorate, do not precipitate bile salts or produce tissue reactions and fibrosis. Intense tissue reaction is a danger if the tubes are contaminated with lint or talc.

Any leakage about an anastomoses is removed by suction.

Summary

1. Cholecystectomy is the most common cause of common duct stricture.

2. Prevention requires respect for normal or anomalous anatomy, meticulous surgical technic, and ideal operative conditions.

3. Adequate preoperative preparation. Decompression procedures may be indicated prior to reconstruction surgery.

4. Surgical exploration of the proximal and distal duct segments is aided by cholangiogram studies.

5. The surgical procedure will vary with the length of the duct above and below the stricture. The following procedures may be utilized.

A. Choledochocholedochostomy

B. Biliary-Entero Anastomoses

1. Choledochoduodenostomy:- Hepaticoduodenostomy
2. Choledochojejunostomy:- Hepaticojejunostomy
   a. Roux-Y Jejunostomy
   b. Jejunojejunostomy
   c. Anastomoses of Jejunum to the hilum of the liver
   d. Intrajejunal anastomoses
   e. Transhepatic intubation
   f. Intrahepatic Cholangiojejunostomy

6. Prolonged use of internal splints are utilized following common duct reconstruction surgery, thereby protecting the anastomoses from contraction of scar tissue.

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Dermatologic Disorders Treated by Oral Triamcinolone

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The steroid, Triamcinolone* (9-alpha-fluoro, 16-alpha Hydroxyprednisolone) has been used and studied for approximately six years. The clinical studies[1,2,3,4,5,6] using oral Triamcinolone revealed highly effective results in the treatment of dermatologic disorders.

It is the purpose of this paper to present our observations on the treatment and management of acute and chronic dermatologic disorders with this agent.

MATERIALS AND METHODS

The Triamcinolone was given orally to 39 patients. The skin problems ranged from common to unusual. There was no attempt to limit therapy to the systemic steroid. Topical and other oral medications were utilized as needed. The duration of therapy varied from one week to seven years.

Appropriate laboratory studies were obtained as warranted.

RESULTS

A summary of results is given in Table 1.

The best results were obtained treating acute conditions that are known for their tendency toward self-limitation. The most impressive results were obtained in the treatment of cases of Dermatitis Venenata due to contact with members of the Rhus family. Twenty-five such cases were treated, all with excellent results. The daily dosage range of Triamcinolone was 16 mgm. gradually reduced to mgm. The duration of therapy was 5 to 21 days.

The two cases of benign mucous membrane pemphigus were treated with prompt improvement noted in the first 2 to 3 weeks. Continuous treatment was required in order to maintain suppression. Periodic increases in the dose were needed to overcome intermittent exacerbations.

Good initial results were obtained in one case of generalized psoriasis but control was lost, even with substantial doses. On withdrawal of this medication, there was a rather distinct increase in the signs requiring diligent therapy with other measures to control them.

TABLE 1

TREATMENT RESULTS WITH ORAL TRIAMCINOLONE IN VARIOUS DERMATOLOGIC DISORDERS

Diagnosis	*Excellent*	*Good*	*Fair*	*Poor*	*Dose Range in mgs. Start*	*Finish*
Atopic Dermatitis		3			24	4
Benign Mucous Membrane Pemphigus		2			32	3
Dermatitis Herpetiformis			1		24	8
Dermatitis Venenata due to Rhus Plants	25				16	4
Erythema Multiforme	1		1		32	2
Neurodermatitis Disseminata		1			24	4
Nummular Dermatitis		2			24	4
Pemphigus Vulgaris		1			48	176
Pityriasis Rosea	1				24	2
Psoriasis Vulgaris		1			24	12
Seborrheic Dermatitis		1			24	4

Atopic dermatitis and neurodermatitis disseminata both showed prompt and gratifying improvement. Unless other measures were used to control the basic condition, recurrences were encountered promptly on even moderate reduction of the dose.

One patient with dermatitis herpetiformis required substantial doses to control the manifestations. Several other physiological effects were encountered, ultimately necessitating cessation of steroid therapy.

A patient with severe pemphigus vulgaris which went on to develop the foliaceous state was improved approximately 80% on massive doses of steroids but was never completely controlled. The patient expired from intercurrent respiratory infection.

REPORT OF CASES

Case 1. A 23-year-old, white, single, male construction worker and cement finisher had no prior history of skin disease.

Approximately seven days before the skin condition developed, the patient had removed his shoes to work in a wet, grassy area. The manifestations began on the dorsum and between the toes, spread up to the ankles and legs, onto the hands, arms and trunk. Itching was very severe. The eruption was characterized by redness, swelling and blisters frequently in a linear arrangement.

Past History: His health had been very good without any knowledge of hypersensitivity.

Physical Examination: The patient was a well-developed, well-nourished, tanned white male. The right forearm, chest, knees and feet revealed many grouped and scattered lesions. These were edematous papules, vesicles and bullae on a red, swollen skin. The lesions were frequently seen in linear arrange-

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ments. The bullae on the feet protruded between the toes. The lower legs were diffusely swollen. The physical findings were otherwise normal.

Diagnosis: Dermatitis Venenata due to Rhus Toxicodendron.

Treatment: Linit Starch bath 15 minutes daily; oily shake lotion with ½% Neomycin Powder; Triamcinolone 8 mgs. every 8 hours for 6 doses, 4 mgs. every 4 hours except the 2 a.m. dose for 10 doses, 4 mgs. every 6 hours for 8 doses, 4 mgs. every 8 hours for 6 doses, 4 mgs. every 12 hours for 4 doses.

Course: After four days of treatment, the blisters and swelling had subsided leaving residual redness and scaling. The itching sensation had almost subsided.

Within two weeks, the patient's skin had returned to practically normal.

Case 2. This 30-year-old, white, male physician had no prior skin diseases. On March 10, 1959 he presented with a two week history of disseminating, discrete but generalized eruption of a severe degree. The lesions were various sized papulo-squamous patches and plaques, tan to red in color. They were so numerous as to appear confluent. The eruption was concentrated at the intertriginous areas.

Diagnosis: Pityriasis Rosea.

Course Under Therapy: His treatment for the first month was mild erythema doses of ultraviolet light, starch baths and Trimeprazine. On March 23, 1959 in the face of increasing lesions and intensification of his symptoms, he was started on Triamcinolone 4 mgs. five times daily. The dose was decreased 4 mgs. every third day. He experienced relief of his symptoms in six hours. There was remarkable clinical improvement in seven days. His skin returned to normal by April 15, 1959.

The steroid tolerance was excellent.

Case 3. A 73-year-old, white, married woman was admitted to the hospital January 28, 1957 for study of recurrent lesions of the nasal and oral mucous membranes, oro-pharynx, conjunctivae and the forearms. The patient had previously enjoyed good general health, free of skin disease. In 1955 she had nasal polyps removed. She used Senna or Agoral® for relief of chronic constipation. Beginning in October of 1955, she experienced periodic appearance of vesicular lesions of the mucous membranes. These lesions would last one day to several weeks with long periods of freedom between attacks. She had been carefully and extensively studied for bacterial and viral infections of the nose and throat and for possible reactions to medicines. All previous studies produced no positive help. The previous treatments included injectable vitamins, repeated smallpox vaccinations and a variety of topical agents.

Weight on admission was 134 pounds; Blood Pressure 142/78. The skin of the upper and lower eyelids showed large xanthelasma. The margins of the eyelids were inflamed. The conjunctivae were pale in color. There was scarring in the lower fornix of each conjunctiva. Scarring had produced cicatrical entropion of the right lower lid. Five or six cilia of the lower right lid repeatedly brushing against the cornea had produced a mild keratitis. There was a fissure at the tip of the right naris. The nasal membranes were inflamed and the erosions were covered with a serous, purulent crusting. On the mucous membranes of the pharynx, hard palate and the lower gingival ridge were seen numerous small bullae, usually on an erythematous base. Some of the bullae arose from normal mucosa. The forearms from immediately above the cubital spaces, extending to the wrists, including medial and lateral aspects, revealed a patchy, erythematous, annular, configurate, eczematous type of change.

The following laboratory studies are reported: complete blood count on several occasions. The original WBC was 10,000 with 10% Eosinophils. A later WBC was 8,000 with 1% Eosinophils. Repeated urinalysis revealed a trace of albumin. Microscopic examination revealed 2-3 RBC and occasional WBC and 8-10 Hyaline and Granular casts per high power field. BUN was 25 and 22 mg. per 100 ml. on separate occasions. On admission, total proteins were 5.7 grams per 100 ml., albumin 3.1 gms. per 100 ml., globulin 2.6 gms. per 100 ml. Electrophoretic studies of the blood April 19, 1957 showed total protein 6.2 grams, albumin 2.9 grams, Alpha 1, 0.4 grams, Alpha 2, 0.6 grams, Beta 1.1 grams, Gamma, 1.1 grams. Serum electrolytes, blood sugar, serum bilirubin, alkaline phosphatase were in normal range.

Biopsy material from the mucous membrane lesions was studied. This revealed no acantholysis. The histologic picture favored that of benign mucous membrane pemphigus. X-ray examination of the chest showed evidence of esophageal cardiospasm. The abdominal and thoracic aorta contained considerable calcification. X-ray of the lumbar spine revealed rather marked narrowing of L 4 and 5 disc space, and considerable sclerotic change about the apposed margins of the vertebrae. Minimal degenerative changes in the bodies of the other lumbar vertebrae were noted. These examinations repeated in 1959 were approximately the same.

Diagnosis: 1. Benign mucous membrane pemphigus.
2. Neurodermatitis disseminata.

Course Under Therapy: On March 28, 1957 the patient was started on oral Prednisone 5 mg. every 6 hours. On April 1, 1957 because of an increase in the clinical signs, the Prednisone was increased to 5 mg. every four hours. With the suppression of the signs, the dose was progressively reduced over a matter of three or four weeks until a maintenance dose of 5 mgs. every 12 hours was used. When the signs increased, the dose was increased to 5 mgs. four times daily. On March 30, 1958 the Triamcinolone 4 mgs. twice daily was used in place of the Prednisone. The base maintenance daily dose has been 2 mgs. Periodically, with an increase in the clinical signs, the dose will be increased to 2 mgs. four times daily. A majority of the flare-ups could be controlled by increasing the total daily dose to 4 or 8 mgs. for one to four days. On one brief occasion, an effort to use the same steroid dose by oral troche, each containing 1 mg. of active ingredient, Triamcinolone, was accompanied by a massive flare-up in a matter of 96 hours.

The eczematous eruption on the arms disappeared with compresses, drying lotions and topical steroid applications during the first six months of observation. This feature has remained absent. There have been some clinical vesicles or erosions constantly present on the mucous membranes except for intervals of one day to two weeks. There is some erythema present in the throat at all times. Occasionally, symptoms are such as to give clinical indications that some of the lesions may be in the upper esophagus. This area has not been visualized. The nasal mucosa of the patient has remained normal. The patient has encountered the usual intercurrent disorders such as respiratory infections and "grippe." Several attacks of arthritis were encountered in spite of the oral steroid therapy. The patient had a steady weight loss from 134 pounds in January 1957 to 110 pounds in March 1965. Her general strength and vitality has been fair but not as great as formerly. The mucous membrane defect in the throat has definitely restricted the volume and type of food she could tolerate. The esophageal cardiospasm has been a restricting factor in the food intake. The contour of the face has definitely become and remains full revealing the classical moon facies. The skin of the exposed areas of the body is quite fragile and will produce ecchymosis very readily. She recognizes urgency and frequency when the steroid dose is increased.

Case 4. A 65-year-old white, retired, male police officer and night watchman was first seen December 31, 1958 with a history of quite rapid development of generalized vesiculobullous eruption of four months duration. Past history reveals persistent hypertension, one episode of coronary occlusion, and a mild cerebral vascular occlusion which cleared without residuum. The past and general history otherwise were noncontributory.

Physical Examination: Height 5'10"; Weight 164 pounds; Blood Pressure 220/100. The patient was pale in appearance but otherwise well preserved and nourished but quite apprehensive. The skin manifestations appeared in the scalp, trunk, extremities, axillary, perineal body fold areas. These were scaly, crusted patches with macular, tan pigmentation. Medium and small sized bullae were noted in these same areas, appearing on the normal skin.

Biopsy of one of the bullae at the time of the first visit revealed changes consistent with pemphigus vulgaris. These studies revealed an intra-epidermal hiatus. Acantholytic epithelial elements were noted. At the patient's request, treatment started as an outpatient. Therapy consisted of Prednisone 5 mg. four times daily for a month and 5 mg. twice daily for two weeks, then two weeks without therapy.

The patient was hospitalized March 3, 1959. The physical findings revealed a grossly uncomfortable white male with a severe burning and stinging of the skin. The lesions were now generalized. They were diffuse, erythematous, edematous scaling process, involving all areas. The other positive physical findings were the increase in the carotid pulsation over the left side of the neck. The left border of the cardiac outline could not be demonstrated by percussion. Heart sounds were distant. There was an occasional premature contraction of the heart. There was a three plus pitting edema of the feet and calves.

Laboratory studies: Blood count normal. Urinalysis showed a protein of three plus. Serum electrolytes in meq/L were sodium 137; potassium 3.8; chlorides 98; CO_2 28 vol. per cent; March 17, 1959: Sodium 129; potassium 2.9; March 24, 1959: Sodium 130; potassium 3.1. Total protein 4.9 grams per 100 c.c.; albumin 2.0 grams per 100 c.c.; globulin 2.9 grams per 100 c.c.; B.U.N. fasting blood sugar, V.D.R.L. were within the range of normal. Chest x-ray showed the heart enlarged in the region of the left ventricle. The aorta was tortuous and elongated and partially calcified. The lungs were clear. EKG revealed a long Q-T wave ratio with evidence of myocardial anoxia.

Diagnosis: Pemphigus Vulgaris.

Course Under Therapy: On March 6, 1959 oral Triamcinolone 12 mg. every six hours was started. Aluminum Hydroxide Gel between meals, phenobarbital and atropine were used daily to relieve mild symptoms of gastro-intestinal irritation. On the fourth hospital day, the patient developed orthopnea, a striking increase in the swelling of the ankles with increase of 10 pound in weight. Digitoxin was started and continued to complete digitalization. On the eighth hospital day, March 10, 1959, the Triamcinolone was increased to 24 mg. every six hours since the clinical signs were improving only very slowly. Chlorothiazide 500 mg. three times daily for three days was given. There was a reduction in weight from 171½ pounds to 146½ pounds, a loss of 25 pounds. On the fifteenth hospital day, March 17, 1959, since the clinical signs were still slow in resolving, the triamcinolone medication was increased to 36 mg. every six hours. The topical measures accompanying the treatment were aerosol Pramoxine Hydrochloride and Prednisolone; mineral oil was used for lubrication, equal parts of zinc oxide and castor oil were applied with dressings over the denuded areas. By March 25, 1959, or the twenty-fourth hospital day, the patient had improved sufficiently to permit discharge to his home. It was estimated he had recovered 80%. There were still many areas of exfoliation. There were fissures on the lower legs with some residual edema and bullae and a few urticarial lesions. Discharge weight 152 pounds; Blood Pressure 230/110.

The patient was then followed on an outpatient basis.

April 1, 1959 the oral Triamcinolone was increased to 44 mgs. every six hours in an effort to improve the residual changes. At this time, the skin of the cheeks showed a great deal of scaliness and erythema. A very fine scaliness was noted on the back, underlying skin was pink. The skin generally had a violaceous appearance but no bullae were seen.

On April 8, 1959 a mild cough developed. Sputum culture was obtained and Candida Albicans and Hemophilus Influenza were found. The patient suddenly expired April 8, 1959. No autopsy was done.

The total dose of steroids received by the patient from March 5, 1959 through April 8, 1959 was 4,112 mg. He received 48 mg. a day between March 5, and March 10, for a total of 240 mgs.; 96 mg. a day between March 10, and March 18, for a total of 758 mgs.; 144 mg. daily from March 18, to April 1, for a total of 1,872 mg.; 176 mg. per day between April 1, and April 8, for a total of 1,232 mgs.

The patient's tolerance of the medicine appeared to be good except for a decrease in the appetite and gaseous eructations.

Other Effects Of Medication

A total of seven patients demonstrated eleven other effects. The most debilitating effect was lethargy and weakness. Four patients recognized these. Three patients had distinct rounding of the face or moon facies. Weight gain was encountered in three patients and rather extensive weight loss in two, one patients lost 24 pounds in approximately two years. Two patients in midyears recognized vasomotor symptoms of hot flashes, stifling and hyperhydrosis. Increased capillary fragility was recognized in two with the development of ecchymosis at areas of moderate trauma. A definite frequency in urination was noted by one patient when the dose was increased. Depression and emotional instability was noted in one patient. Anorexia was encountered in one patient. Leg cramps of relatively distinct type were noted in two, one on short duration therapy and the other after longer period of treatment. Concurrent development of acne in one adolescent male was encountered and seemed to be more pronounced when the dosage schedule was high. This patient required continuous treatment for more than a year.

Comment

The steroid, Triamcinolone, after several years of trial, appears to have retained its anticipated effectiveness in the treatment of dermatologic conditions. It compares favorably with other steroid derivatives. It appears to permit the development of similar and equal number of other effects.

A careful selection of the cases to be so treated is highly essential. It is entirely possible that a patient may become dependent upon this medication, and it is most difficult to wean them when the symptoms and signs tend to recur.

The use of other appropriate oral and topical treatments will frequently shorten the duration of the patient's problem.

It is highly essential that persons under steroid therapy be carefully observed at specific intervals so that the appearance or increased severity of any other physiologic effects can be noted and dealt with.

Certainly in dermatoses of acute severe type where the condition is anticipated to be of short duration, this therapy is extremely valuable.

This agent unquestionably has proven its value in dermatologic conditions which would have shortened life. Judiciously used in more stubborn chronic recurrent

conditions, it has been of inestimable value in controlling severe symptoms and disability.

Summary

Thirty-nine patients with various acute and chronic dermatologic disorders were treated with oral Triamcinolone. The overall results of therapy were excellent to good. The limitations and course under therapy are discussed.

Generic and Trade Names

Aerosol Pramoxine Hydrochloride = Tronothane®
Aerosol Prednisolone = Meti-Derm®
Trimeprazine = Temaril® Spansules®
Chlorothiazide = Diuril®

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COMMON DUCT RECONSTRUCTION OUTLINE FOR TREATMENT — *Continued from page* 161

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142 Joralemon Street, Brooklyn, New York

Topical Triamcinolone Acetonide as an Adjunct to Dermatologic Therapy

Arthur H. Flower, Jr., M.D.†

Numerous studies[1,2,3,4] have established the value of topical steroids in the practice of dermatologic medicine. This presentation is a discussion of the results of practical application of one of the topical steroids in the course of managing cutaneous disorders. The preparation under consideration is Triamcinolone Acetonide.* The chemical characteristics and pharmacologic activity of this agent have been studied thoroughly by Borman and Lerner.[5]

Selection Of Patients

The cases were selected on the basis of the probability of their responding to topical steroid therapy.

All were treated in standard fashion. The various members of the therapeutic armamentarium were employed in their proper sequence in an effort to provide the patient relief and eventual recovery from the disorder. These included baths, compresses, antibacterial agents topically and orally, sedation or ataratics and physical therapy when appropriate. The topical steroid was introduced into the regimen when it was felt it could be used to the greatest advantage to improve the overall clinical problem.

Materials Used

The active ingredient was 0.1% Triamcinolone Acetonide. The vehicles were several: cream, oleaginous ointment and lotion base. Triamcinolone Acetonide was used alone in these vehicles and also coupled with Neomycin, Gramicidin and Nystatin. The patients were given detailed instruction in the proper method of application of these materials.

Results

A total of 59 patients were treated. The variety of conditions encountered and results obtained are outlined in detail in the accompanying Table 1.

Fifty-two of the patients experienced excellent to good results. There were five fair results and two failures. In most instances, the response to medication was quite prompt, almost dramatic at times.

Comments

The response to therapy in this group of cases was uniformly good. It was found that the medication was most effective when the skin had been brought to a state of improvement by the reduction of the edema and the elimination of the bacterial flora to as great an extent as possible. In those instances where the total picture of the patient was so arranged, he usually went on to complete healing and remained in a stable state. In those problems in which the total picture was unstable, it was not uncommon to encounter recurrence. The reuse of the topical triamcinolone Acetonide in most instances promptly restored the former state of improvement. The rapidity of response was striking. In many instances within 24 to 48 hours, there was remarkable improvement. It was usually necessary to treat the patient to the point that the skin returned to as near visual normality as possible. When therapy was withdrawn earlier than this, recurrences were more common. If the steroid preparation was committed to the therapeutic regimen too early, the disease failed to improve to the degree noted above. The condition would improve to some degree but seemed to hit a plateau and fail to go beyond that. Addition of the antibacterial or antimycotic ingredients did have a decided benefit and in some instances these agents were able to do the job entirely without the above noted prior preparation of the skin. However, if the degree of infection and the amount of edema were marked, they also would fail to bring about complete resolution.

The skin problems where only fair results were achieved included four instances of quite severe atopic dermatitis where it was difficult to cope with the total problem, and one instance of keratosis follicularis. The topical steroid was used primarily to aid in reducing itching in the latter instance, and did so. Since the basic problem was not improved, there was a tendency for recurrence of symptoms. The first case of failure was one of neurotic excoriations. The compulsion for attacking the skin was so strong that the anti-inflammatory effect of the steroid had little influence on the process. The other failure was an instance of proved dermatophytosis due to epidermophyton floccosum. While there was temporary suppression of the inflammation, the clinical signs recurred promptly on discontinuance of therapy.

There were no noted systemic or local adverse effects

*Supplied as Kenalog preparations by John T. Groel, M.D. Associate Clinical Research Director, Squibb Institute for Medical Research, New Brunswick, New Jersey.

†Associate Professor of Clinical Dermatology, College of Medicine, University of Vermont.

TABLE 1

Treatment Results of Topical Triamcinolone Acetonide in Various Dermatoses

DIAGNOSIS	*Excellent*	*Good*	*Fair*	*Poor*	*Failure*	*COMMENTS*
Neurotic Excoriations					1	Very little help in control of signs and symptoms.
Atopic Dermatitis	2	5	4			Not effective when infected.
(E. Floccosum) Dermatophytosis					1	Signs could be temporarily suppressed but returned on stopping therapy.
Dermatitis Herpetiformis		1				Mildly effective for symptom control.
Dermatitis Venenata	6					Effective in early stages or less severe states.
(Dariers Disease) Keratosis Follicularis			1			Used primarily to control itching.
Neurodermatitis Disseminata	6	1				Very helpful in controlling signs and symptoms.
Nummular Dermatitis		4				Continuous applications of medication needed to control signs.
Psoriasis Vulgaris	2					Effective in the fold areas and macular lesions.
Recurrent vesicular Eruption hands	1	1				Frequently required continuing use to maintain control.
Seborrheic Dermatitis	15	8				Best responses were superficial uninfected lesions.
Total	32	20	5		2	

of this agent. In the event topical steroid was used in a field where bacteria were prone to occur, a severe flare-up of the bacterial component was encountered on several occasions. The careful instructions for the frugal use of the agent undoubtedly permitted very little of the medicine to be absorbed.

The various bases available for the Triamcinolone Acetonide gave a wider applicability of the agent. The base itself had very little effect upon efficacy. The variety of available bases improved patient acceptance of the medication.

Summary

Fifty-nine selected dermatologic cases of varied etiology were studied. Topical Triamcinolone Acetonide preparations were utilized as an adjunct to other therapeutic measures. They were committed to the treatment regimens when it was felt they would produce the greatest response. There were 52 good to excellent and 5 fair results and 2 failures.

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Maine Medical Association

STANDING COMMITTEES 1965-1966

Standing Committees for 1965-1966 as proposed by the Nominating Committee and approved at the Second Meeting of the House of Delegates of the Maine Medical Association at Rockland, Maine, June 13, 1965.

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President
Maine Medical Association
1965 - 1966



JOHN F. DOUGHERTY, M.D.

JOHN F. DOUGHERTY, M.D. of Bath, Maine assumed his duties as President of the Maine Medical Association on June 14, 1965 at the 112th Annual Session banquet. Dr. Dougherty served as Councilor for the Third District from 1959 to 1965, the last year as Council Chairman and as President-Elect from 1964 to 1965.

Dr. Dougherty was born in Bath, Maine on November 28, 1905, the son of Daniel T. and Catherine G. Dougherty. He attended Georgetown University, the University of Maine, Bates College and received his medical degree from Boston University School of Medicine in 1939. He did postgraduate study at Boston City Hospital and St. Elizabeths Hospital from 1939 to 1941 and served in the U.S. Army Medical Corp from 1941 to 1946.

Dr. Dougherty is a member of the American Medical Association, the Maine Medical Association and the Lincoln-Sagadahoc County Medical Society.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Review of Health and Welfare Legislation

DEAN FISHER, M.D.*

With the exception of items of legislation passed under a so-called "emergency preamble," all legislation goes into effect 90 days after the Legislature adjourns, unless some other date is specified. The date of effectiveness of most legislation will, therefore, be September 3, 1965.

The major appropriation acts go into effect on July 1, 1965, the beginning of the fiscal year.

We do not yet know the final fate, or final form, of some legislative items that were processed the last few hours of the session. Therefore, all legislative items cannot be reviewed at this time. However, we shall be receiving official copies of such acts very soon.

The 102nd Maine Legislature considered a great variety of proposals that were of interest to the Department of Health and Welfare, and of varying degrees of interest to the medical profession. The items which I shall discuss briefly are not the only ones in which the medical profession may be interested. For example, legislation related to the Departments of Labor, Mental Health and Corrections, Education, Agriculture, and others, might properly be included in any comprehensive review, but I am not yet sufficiently familiar with such acts as to be able to interpret them adequately.

To us, the appropriation acts are the most important documents. They provide not only program maintenance funds, but they can also be the means for major program expansion, re-direction, or revision.

Capital appropriations provide for extensive additions of equipment to our laboratories, and new and more adequate quarters for the entire department. The additional space is badly needed. The laboratory equipment will permit offering a variety of services not now available.

The so-called current services appropriation act provides quite well for the continuation of existing programs, and makes possible some minor adjustments.

The supplemental appropriation act provides for 56 new positions, 48 of which are in the public health nurse or social worker series. A most significant appropriation of $492,000 per year is made for improvements in foster home care, education, medical care, etc. for committed children. $600,000 per year is appropriated for additional medical care services or improvements in rates of payment for care in public assistance and hospital aid. Our requests had been for considerably larger amounts for both general and specific medical care funds. However, the appropriations made should provide for important changes.

At least five items of interest were assigned to the Legislative Research Committee for interim study, as follows:

(1) Study of need for air pollution legislation.
(2) Study of guardianship or other protective provisions that may need to be made for those unable to manage their own affairs, such as the mentally retarded.
(3) Study of nursing homes, boarding homes, and children's foster homes.
(4) Study of the adequacy of the program of rehabilitation for the blind.
(5) Study of the program for alcoholic rehabilitation.

Provisions for general salary adjustments were made, and this may help in recruiting and retaining capable staff.

Some specific legislative items of interest are the following:

(1) The Department is authorized to require hospitals to do PKU testing on newborns.
(2) The bills to license private laboratories were not passed.
(3) The ownership of the tuberculosis wing on the Fort Fairfield community hospital was transferred to that hospital. Parts of the wing will be used for tuberculosis outpatient services and for community mental health services.

Continued on Page 174

*Commissioner

Maine Heart Association Notes
116 State Street, Augusta, Maine

The Rationale and Methods For Combatting Hypercapnia In Cor Pulmonale

"The major therapeutic challenge in these individuals lies in improving oxygen saturation and simultaneously reducing the level of carbon dioxide. This can be achieved in a number of different ways. Foremost - - - is the use of antibiotics to suppress the exudates and secretions - - - which clog the tracheobronchial tree. This, coupled with tracheal suction and adequate hydration, is extremely effective in furthering alveolar ventilation. The systematic use of vaporized bronchodilators - - - reduces mucosal edema and bronchial obstruction - - -. Positive pressure breathing using moist air interchangeably with bronchodilators is very effective in hyperventilating these subjects and should be employed as continuously as possible. - - - smoking, which is a powerful irritant that results in bronchorrhea, is contraindicated. Similarly, the suppression of cough by drugs or - - - narcotics, tranquilizers, or barbiturates, with their attendant respiratory depressant effect, cannot be condoned.

"Restriction of activity cannot be overemphasized as a major therapeutic measure in the care of these patients, not only in their acute episode but also while they are convalescing. - - - For these individuals who are laboring toward effective gas exchange, the additional burden of work should be removed.

"The best means available at this time for judging whether any of these therapies is successful is a determination of the arterial blood oxygen saturation and carbon-dioxide tension."

Submitted by Jacob B. Dana, M.D.

Reference: Ferrer, M. I., and Harvey, R. M. American Heart Journal, Volume 69, pages 419-421, 1965.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

The Leukemia Cell and The Virus

NEW YORK, N. Y. — An animal leukemia virus has proved to be enormously resistant to x-rays, although the leukemia cells which harbor it are extremely sensitive.

This new finding may explain why leukemic animals and humans often obtain a remarkable but temporary remission after x-ray therapy. The leukemic cells die, but the invincible virus persists, infects new cells and causes a recurrence of the disease.

The American Cancer Society reported recently on these observations by a team of scientists working with Dr. Ludwik Gross at the Cancer Research Unit of the Bronx Veterans Administration Hospital.

The scientists collected common and very deadly leukemia viruses (passage A, or Gross) from leukemic rats, sealed them in ampoules and exposed them to x-rays of various high intensities. The radiation dosages ranged from 100,000 to 1,000,000 roentgen units, delivered by fuel elements from a nuclear reactor.

The irradiated viruses then were injected into newborn rats. The only viruses halfway inactivated were those exposed to 1,000,000 roentgen units; and, even then, five of the 11 rats inoculated with them came down with the disease a few months later.

Of 38 rats given viruses irradiated with up to 750,000 roentgen units, 37 developed leukemia when they were little more than four months old. Only one escaped the disease.

Non-irradiated virus caused leukemia in all injected rats.

The scientists then turned to experiments with leukemic cells removed from sick mice which had been injected with the virus. A dose as low as 5,000 roentgen units inactivated the cells so thoroughly that they did not cause any leukemia when injected into young adult mice.

Dr. Gross first isolated the virus from "spontaneously" leukemic mice 14 years ago. He found that the virus caused leukemia not only in young mice but in newborn rats as well. It could be harvested from both animal species, and it grew in virulence as time passed.

Leukemia, which is cancer of the blood-forming organs, often involves not only the blood, but also the thymus, spleen, kidney, liver and the bone marrow. It is closely related to solid but disseminated cancers of lymphatic tissues, such as lymphosarcoma and Hodgkin's disease.

Over the years, Dr. Gross has found his virus associated with quite a few kinds of animal leukemia — spontaneous or induced by either radiation or chemicals — and some lymphatic cancers.

He has now raised the question as to whether all mouse and rat leukemias may not stem from virus A — the original virus A, its mutant forms and forms resulting from adaptation to new species and strains of animals.

From American Cancer Society News Service Release

LOMOTIL—*Pharmacologic Activity.*

The significant pharmacologic actions of Lomotil are summarized as follows:

Evidence indicates that Lomotil acts directly on the intestinal musculature to inhibit excess peristalsis.

Lomotil is not known to inhibit nonpropulsive intestinal movements.

Roentgenograms demonstrate that this activity occurs within two hours after oral administration and persists for at least six hours.

Comparative studies in the rat show Lomotil to be more effective in inhibiting fecal excretion than either codeine or morphine.

Analgesic, anticholinergic, mydriatic and gastric secretory effects have not been significant.

Reduction of propulsive motility with Lomotil relieves spasm and cramping, allows physiologic absorption of fluid and reduces frequency of evacuations to provide prompt, symptomatic control of virtually all diarrheas.

LOMOTIL® tablets liquid

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride .....2.5 mg.
(Warning: May be habit forming)
atropine sulfate .....0.025 mg.

slows propulsion · relieves distress · stops diarrhea



Precautions: Lomotil is an exempt narcotic preparation of very low addictive potential: more than three million prescriptions have now been written for Lomotil. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

Dosage: For full therapeutic effect – Rx full therapeutic dosage. The recommended initial daily dosages, *given in divided doses,* until diarrhea is controlled, are:

Children:
3 to 6 months—3 mg. (½ tsp.* t.i.d.)
6 to 12 months—4 mg. (½ tsp. q.i.d.)
1 to 2 years—5 mg. (½ tsp. 5 times daily)
2 to 5 years—6 mg. (1 tsp. t.i.d.)
5 to 8 years—8 mg. (1 tsp. q.i.d.)
8 to 12 years—10 mg. (1 tsp. 5 times daily)

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) **Based on 4 cc. per teaspoonful.*

Maintenance dosage may be as low as one fourth the therapeutic dose.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdosage.

SEARLE *Research in the Service of Medicine*

DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page* 170

(4) An act providing for reporting by physicians of physically abused children was passed in a relatively model statute form.
(5) The residence requirement in public assistance was eliminated.
(6) A statutory definition of a "nursing home" was passed, but probably will need considerable interpretation.
(7) The statutory maximums on public assistance grants were removed, and an eligible child in Aid to Dependent Children re-defined to encourage continuation in school. The so-called "relatives' responsibility law" in public assistance was further liberalized.
(8) Licensing authority for day care centers was granted.
(9) A service to locate deserting parents liable for support of dependents was authorized.
(10) Authority to enter into the New England Welfare Compact was extended.

There is no pretence that the above is a complete listing of legislative items.

There is such a close interrelationship between many of our activities and federal acts that the total effect of much state legislation cannot be known until congressional actions have been completed.

It seems likely that circumstances will be such that an increasingly close working relationship between the medical profession and this department will be advantageous to both, if we are to develop realistic solutions to some of the complex problems that may be of mutual concern in the foreseeable future.

STANDING COMMITTEES 1965-1966 — *Continued from Page* 168

Carlton E. Swett, M.D., Fairview Hospital, Skowhegan (3 yrs.) – (Somerset)
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John Kazutow, M.D., Box 113, Columbia Falls (3 yrs.) – (Washington)
Roger J. P. Robert, M.D., 331 Main St., Saco (2 yrs.) – (York)

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Maine Chapter, American College of Surgeons – John F. Reynolds, M.D., 216 Main St., Waterville
Maine Trauma Committee – John A. Woodcock, M.D., 35 Second St., Bangor
Ear, Nose and Throat Group – John E. Whitworth, M.D., 336 Mt. Hope Ave., Bangor
Maine Society of Pathologists – Franklin F. Ferguson, M.D., 22 Bramhall St., Portland
Neurology and Neurosurgery Group – Daniel A. Rock, M.D., 477 Main St., Lewiston

Delegate and Alternate Delegate to A.M.A.

(January 1, 1965 to January 1, 1967)

Delegate – Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville
Alternate Delegate – George W. Wood, III, M.D., 156 No. Main St., Brewer

County Society Notes

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on May 18, 1965.

The President, Carl R. Griffin, Jr., M.D., called the meeting to order.

A letter from Raymond W. Farnham, principal of Morse High School in Bath, was read in which he requests copies of *Today's Health* magazine for the high school. It was voted that the secretary write the American Medical Association for rates of subscriptions to this magazine and proceed to order copies for all the secondary schools in the two counties, up to a total cost of fifty dollars.

The proposed Maine Medical Association budget for 1966 was discussed and the delegates authorized to approve this budget at the annual meeting in June.

Francis A. Winchenbach, M.D. reported that the Council of the Maine Medical Association has voted to accept the principles of coding and relative value scales of the Physicians Service Index for Blue Shield. The society voted its approval of this action. He further reported that a ceiling for service benefits of Federal Employees Insurance plans had been accepted at $5,000 for an individual and $7,500 for a family. The society voted approval of this action.

GEORGE W. BOSTWICK, M.D.
Secretary

CUMBERLAND

The Cumberland County Medical Society met at the Holiday Inn in Portland, Maine on May 20, 1965 with seventy-two members and guests attending.

Following a social hour and dinner, the President, Alvin A. Morrison, M.D., introduced Mr. Robert Harris of the Public Service Division of the Southern Maine Life Underwriters Association. Mr. Harris reviewed the accomplishments of the Medic-Alert Program during 1964 and outlined the proposed objectives during 1965. It was voted that the society contribute $50.00 to the Medic-Alert Program for 1965.

Dr. Morrison announced that the American Academy of General Practice was giving up its group professional liability insurance and that Dr. Hanley stated that information relative to coverage would be sent to members from the Maine Medical Association office on request.

Maurice Van Lonkhuyzen, M.D. discussed an outbreak of food poisoning in the local area. He also announced the forthcoming immunization clinics and requested that dog bites be reported to the Health Department.

Albert Aranson, M.D. reported on a recent meeting of the Health Insurance Committee and William C. Burrage, M.D. discussed Veterans' Affairs.

STANLEY B. SYLVESTER, M.D.
Secretary

AROOSTOOK

A meeting of the Aroostook County Medical Society was held on May 26, 1965 at the Prestile Motel in Presque Isle, Maine.

The following officers were elected for 1965:

President, R. Paul Johnson, M.D., Fort Kent
Vice-President, Samuel Rideout, M.D., Fort Fairfield
Secretary, George J. Harrison, M.D., Houlton
Treasurer, Clyde I. Swett, M.D., Island Falls

Delegates to the Maine Medical Association House of Delegates: Drs. Melvin R. Aungst, Fort Kent, John B. Madigan, Houlton and Raymond G. Giberson, Presque Isle.

Alternates: Drs. Rodrigue J. Albert, Fort Kent, George F. Higgins, Presque Isle and H. Douglas Collins, Caribou

Board of Censors: Drs. Eugene G. Gormley, Houlton, Melvin R. Aungst and Gerald H. Donahue, Presque Isle

GEORGE J. HARRISON, M.D.
Secretary

YORK

Twenty-four members and four guests were present at the meeting of the York County Medical Society which was held at the Notre Dame Hospital in Biddeford, Maine on June 9, 1965.

Following a social hour and dinner, the meeting was called to order by the President, Alvin A. Hoffman, M.D. Two visiting lawyers Daniel E. Crowley and Simon Spill, both of Biddeford, were introduced and talked about a joint meeting of doctors and lawyers. Drs. Alvin A. Hoffman, Kenneth E. Leigh, Leopold A. Viger and Charles W. Kinghorn were appointed to meet with a committee of lawyers to set a date and place.

Paul S. Hill, Jr., M.D., Councilor for the First District, gave a talk on Maine Medical Association matters.

Harold L. Osher, M.D. of Portland, Maine gave a very interesting and instructive talk on heart conditions.

Secretary
CHARLES W. KINGHORN, M.D.

Letter to the Editor

To The Editor:

The publication of a paper on the use of triamcinolone in skin disorders, which appears in this issue of The Journal, does not appear to be a very critical study. A review article bringing the extensive clinical experience on this subject up to date would be very interesting and timely; but the paper on topical therapy does not serve that purpose. I do not think it is very enlightening simply to itemize a group of cases in which topical steroids were used. There is no attempt to compare triamcinolone with other steroids, or to demonstrate any general principles regarding the use of topical steroids.

In the paper on oral treatment, also appearing in this issue, the details of the laboratory findings are mostly irrelevant. A medical workup for a steroid-treated patient should put the emphasis on tuberculosis, blood pressure, blood sugar, etc. There is at least one case which may involve an error. He states that the response to treatment was good, for his case of pemphigus vulgaris. However the patient went on to die, presumedly of a pulmonary infection. The accepted treatment today, according to Walter Lever[1] and others, is to use very

Continued on page 176

LETTER TO THE EDITOR — *Continued from page* 175

large initial, completely suppressive doses of steroids, in an effort to prevent such a death.

Steroids are no longer empirically used in pityriasis rosea, neurotic excoriations, and dermatophytosis; and there are no general conclusions identifying the skin diseases in which these drugs are indicated.

Topical and systemic steroids are being used in great quantities for skin diseases, and possibly not always with discretion. A paper which presented some of the current principles of treatment would help put this busy area of medicine in proper perspective.

SAMSON FISHER, M.D.
Waterville, Maine

1. Lever, W. F. and White, H.: Treatment of pemphigus with corticosteroids. Arch. Derm., 87:12, 1963.

This letter and comment was solicited by the Editorial Board

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General Practitioners 45 or under to assist Attending Staff and General Practice Residents in 260 bed general hospital. Annual appointment preferred. $15,000-$17,500 depending on training and experience. Contact Medical Director, San Luis Obispo General Hospital, San Luis Obispo, California. Phone: 805-543-1500.

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Suppurative Cholangitis with Multiple Liver Abscess Formation Due to Aerobacter Aerogenes and Streptococcus Viridans

Successful Treatment with Massive Doses of Intravenous Penicillin†

Fennell P. Turner, M.D.* and Robert L. Ohler, M.D.**

The development of multiple liver abscesses has always been associated with a grave prognosis, and prior to the advent of antibiotics the outcome was almost invariably fatal.[1,2] We feel that the cure of such a patient by means of cholecystostomy, drainage of multiple isolated abscess pockets, and massive intravenous penicillin is of sufficient interest to warrant publication of this case report.

Case Report

This 50-year-old man was admitted to the hospital on 3/21/65 with an acute attack of fever, chills, jaundice, intermittent nausea and vomiting and right upper quadrant distress of two weeks' duration. His past history was essentially negative with the exception of a mild attack of jaundice 21 years before while on a troopship en route to the European Theatre of Operations. For a period of approximately one year prior to admission there had been complaints of vague epigastric distress and sour stomach, associated with the intake of fatty foods. For six weeks prior to admission the symptoms had increased in severity, and there had been associated anorexia and weight loss. When first seen by a physician these symptoms had been sufficiently vague as to preclude a clear-cut diagnosis. Six days later, because of an increase in severity of symptoms, the patient was hospitalized. A provisional diagnosis of infectious hepatitis was made, and the patient was transferred to the VA Hospital, Togus, Maine.

On examination the patient was a moderately obese, acutely ill, jaundiced male of large frame lying quietly in bed and showing evidence of mental confusion. His temperature was 102.2; pulse, 144; blood pressure, 120/80. There was a cataract in the left eye, and the sclerae were icteric. There were scattered rales in the right lung base, which cleared on coughing. Heart was normal. The abdomen was obese and slightly distended, but peristalsis was normal. There was vague tenderness in the right upper quadrant without evidence of spasm. The liver was palpable three fingerbreadths below the right costal margin. There was also evidence of residuals of shrapnel wounds, right lower chest, right upper abdomen, right forearm and hand. There was stasis dermatitis, right leg.

Laboratory studies revealed the urine to contain bile. Hematocrit was 42%; hemoglobin, 12.8 gm.; white count 17,700, polys 70%; sedimentation rate, 76 mm. per hour; serum bilirubin was 8.75 mg.; alkaline phosphatase, 12 Bodansky units; SGOT, 95; prothrombin, 83%; total proteins, 5.9 gm.; albumin 3.1 gm.; globulin, 2.8 gm.; cephalin flocculation, 3 plus; thymol turbidity, 4 units; the serum amylase was at the upper limits of normal; stool guaiac was negative. Examination of urine and blood for spirochetes was negative. Serology, negative. An x-ray of the chest showed elevation of the right leaf of the diaphragm. A flat plate of the abdomen revealed several slightly distended loops of small bowel. In addition, there were several small metallic densities in the left upper quadrant, residual of shrapnel wounds.

Our initial impression was that the patient either had infectious hepatitis or obstructive jaundice associated with cholelithiasis, cholecystitis, and cholangitis. Continued observation in the hospital showed evidence of increasing severity of jaundice associated with further elevation of the alkaline phosphatase and white blood count. The fever was septic and picket-fence in type (rectal temperature reaching 102°-103°F. daily), and there was increasing size and tenderness of the liver. An additional finding was gradual drop in hematocrit from an admission value of 42% to 31% without any evidence of external bleeding. Blood cultures were negative. During this period the patient was intermittently confused and delirious, and he remained quite refractory to examination and questioning. As the patient at no time exhibited classical evidence of peritonitis or peritoneal irritation, the diagnosis remained in doubt although it seemed likely that he had a surgical illness associated with cholangitis rather than infectious hepatitis. A diagnostic peritoneal tap was carried out, and no fluid was obtained.

On 3/28/65 he was started on wide-spectrum antibiotic therapy (Chloramphenicol, 3.0 gm. daily), and on 3/31/65 when temperature reached 105°F., he was started on large intramuscular doses of aqueous penicillin (Potassium Penicillin-G, 1 million units q. 2 h.) in preparation for ex-

†From the Veterans Administration Center, Togus, Maine.
*Chief, Surgical Service.
**Chief, Medical Service.

ploratory laparotomy, which was carried out the following day (4/1/65). At operation the liver was found to be swollen, of a dark purplish-gray color; and the surface was studded with many yellowish areas of fluctuation. These areas varied in size from several mm. to 2 cm. in diameter. The liver itself was densely adherent to the anterior chest wall with both old and recent inflammatory adhesions. The omentum was also strongly adherent to the gallbladder and to the under surface of the liver. The gallbladder was small, contracted, and appeared acutely and chronically inflamed. The lumen contained a cheesy exudate without stones. There was no evidence of peritonitis or subhepatic abscess. While exploration was being carried out, lateral to the liver a gush of pus was observed. This was found to be due to drainage from an area of necrosis in the lateral aspect of the right lobe of the liver. The four largest areas of fluctuation on the under surface of the right lobe of the liver were then incised and opened widely with a Kelly clamp. Several hundred cc. of thick, creamy yellow pus were obtained from each of these fluctuant areas. The gallbladder was then dissected free and opened, and the caseous material removed with stone forceps. No stones were found. The common duct was observed to be of normal size without palpable evidence of tumor or stones. A No. 20 Pezzer catheter was placed within the fundus of the gallbladder. Morison's pouch was drained with two rubber tubes and two Penrose drains. Cultures were positive from both the liver abscesses and from the gallbladder for Aerobacter aerogenes and alpha hemolytic Streptococcus, sensitive by disk method to Chloramphenicol, Colistin and Tetracycline. Using standard sensitivity disks, the bacillus was found to be penicillin-resistant, but by a tube-dilution technique it was found that it was inhibited by penicillin in a concentration of 500 units per ml. In view of this finding it was elected to institute massive doses of intravenous penicillin. According to the method of Weinstein,[3] on 4/2/65 he received 48 million units intravenously of Potassium Penicillin-G.

For the first two days after surgery there was a satisfactory drop in temperature, and there was evidence of drainage of bile via the Levin tube and also through the cholecystostomy and right-upper-quadrant drainage tubes. Urinary output, which had been satisfactory prior to surgery, was 300 ml., and the BUN, which had been 23 mg. %, had risen to 84 mg. %. As it was felt that the patient might be developing renal failure, hemodialysis with the Travenol twin-coil artificial kidney was carried out. The blood urea nitrogen the next day was 68 mg. %, and his urinary output was 1,160 ml. Serum creatinine was 6.4 mg. On the day before dialysis it was also seen that the patient was exhibiting diffuse muscular twitchings, purposeless movements resembling Sydenham's chorea, and neuromuscular irritability evidence by the Chvostek sign. This did not respond to intravenous calcium gluconate or magnesium sulfate. Following the hemodialysis the patient was alert, but the generalized muscular twitchings and irritability continued. As the serum potassium was slightly elevated, sodium penicillin* was obtained as in the doses being administered it was apparent that there was a real danger of administration of dangerous amount of potassium. As the muscular twitching did not respond to dialysis or Mannitol intravenously, it was considered that this, together with other evidence of central nervous system irritability, was due to the massive doses of intravenous penicillin.[3] The penicillin dosage was then decreased. By 4/6/65, his fifth postoperative day, the patient's jaundice was less severe. The BUN, however, remained elevated and in fact continued to rise following operation to heights of 140 mg. % and 160 mg. % for at least two weeks following operation. The creatinine also remained elevated at between 4 and 6 mg. %. Serum bilirubin gradually fell along with the alkaline phosphatase. Urinary output, with the exception of the relative oliguria of the first two postoperative days, remained at satisfactory levels with an output of more than 1,000 cc. per day, suggesting renal tubular dysfunction.

As a consequence of the massive antibiotic therapy, cultures for Streptococcus viridans and Aerobacter aerogenes remained negative after the fourth postoperative day. In their place a pure growth of Candida albicans could now be cultured through both the gallbladder drainage tube and the sump drains in the right upper quadrant. On the sixth postoperative day sodium penicillin therapy was decreased to 3 million units every six hours and by the seventh day it was decreased to 2 million units every six hours. Supportive therapy with occasional transfusion of whole blood as well as serum albumin was carried out. The patient showed continued moderate pyrexia, mental confusion, and at times delirium; however, by the ninth postoperative day the patient had become mentally clear; his temperature was low grade; the appetite had begun to improve; and there was evidence of further diuresis. The gallbladder tube at this point was draining clear white bile. By 4/25/65 the patient was switched to intramuscular and oral administration, and intravenous penicillin therapy was discontinued. On the 26th postoperative day there was spontaneous drainage of several hundred cc. of creamy pus from a small opening in the upper end of the exploratory incision. Surprisingly, a culture of this drainage was once again found to be positive for Streptococcus and for Aerobacter aerogenes. That this particular abscess pocket was well localized, however, was shown by the rapid lysis of fever following drainage and irrigation. Some time later there was evidence of communication between this mid-line wound abscess and the stab wound drainage site of the cholecystostomy tube in the right upper quadrant. Through-and-through irrigation was established with saline and later with Dakin's solution. This abscess pocket eventually disappeared, and at the present time there is only a small amount of drainage daily from the cholecystostomy drainage wound. At the present moment all of his chemical and hematological values are within normal limits inc'uding the BUN; he is fully ambulatory; and other than vitamin therapy, the only drug he is receiving is a maintenance dose of Gantrisin® as a urinary antiseptic because of the prolonged period of indwelling catheterization.

A 1:64 dilution of serum drawn on 4/10/65 (when the blood urea was 140 mg.) inhibited an E. coli test organism known to require 500 units of penicillin per ml. for its inhibition. At this time the patient was receiving 2 million units of penicillin intravenously every six hours. There were no nervous system symptoms at this time.

Discussion

Pyogenic liver abscesses may follow suppurative cholangitis or pylephlebitis. It has been pointed out that the diagnosis of suppurative cholangitis is not a difficult one to make when the characteristic symptoms of chills, fever, and right-upper-quadrant pain occur in a patient with a history of long-standing biliary tract disease.[4] However, it is also recognized that when a patient is first observed in the advanced stage of the disease the diagnosis then may sometimes be extremely difficult. Because of the mental confusion and extreme refractoriness of this particular patient to history taking and to physical examination, there was some delay in establishing a diagnosis. Although gallbladder disease and cholangitis were thought of, it was felt necessary to rule out infectious hepatitis and other causes of jaundice before the proper diagnosis could be established. Suppurative cholangitis due to various organisms has been

*Sodium Penicillin obtained through the courtesy of Mr. Ernest Donagan, E. R. Squibb Co., Portland, Maine.

recognized for a long time.[1,5] The organisms are almost invariably enteric in origin; and while on occasion an organism is grown in pure culture, a mixed flora is usually obtained. Various intestinal streptococci, both aerobic and anaerobic, have often been incriminated. Coliform organisms are also frequently found. The appearance of primary suppuration of the liver or of pyogenic liver abscesses due to the Klebsiella-Aerobacter aerogenes group or what has sometimes been called Friedlander's bacillus has been described for many years, and the difficulty in treatment has been long recognized.[6,7,8] Whereas at one time it was thought that cholangitic abscesses can occur de novo, it is now recognized that liver infection of this type is usually associated with biliary tract obstruction either from calculi, stricture, or bile duct and ampullary carcinoma. More rarely congenital abnormalities of the biliary tracts have been found. In the Orient cholangiohepatitis has been found to be one of the common causes of acute abdominal emergency. Frequently, but not always, such cholangiohepatitis has been found to be associated with an infestation of Clonorchis Sinensis or other worms or parasites.[5,9] These organisms, living or dead, become surrounded by inflammatory debris or multiple small calculi resulting in multiple areas of obstruction in the smaller biliary radicals throughout the liver. Streptococcus fecalis and B. coli are frequently associated bacterial organisms. Years ago a condition called cholangitis lenta was described, the term being devised to imply a similarity with subacute bacterial endocarditis.[10] In these instances a primary infection with Streptococcus viridans was found. These patients typically exhibited chills, fever, bacteremia, severe anemia, and cachexia. Jaundice and hepatomegaly were inconstant findings of grave prognostic significance. In more recent years doubt has been cast on the specificity of this particular entity since it has been shown that many organisms are capable of producing the same clinical picture, and as an underlying stone or malignancy or other cause of biliary tract obstruction has often been an associated finding.

As in suppurative cholangitis the bile ducts are the route of infection, the distribution of the infectious process corresponds in general to the architecture of the bile-duct system with the subsequent development of multiple suppurative foci. In such cases the liver may sometimes double in size and weight. Perihepatitis is also common, and dense inflammatory adhesions develop between the liver, diaphragm and abdominal wall. As the infection becomes more severe, coagulation necrosis takes place, suppuration ensues, and multiple abscesses appear. The areas of abscess formation are characteristically pale yellow in color contrasting markedly with the deep purplish color of the uninvolved and swollen liver tissue. The areas of necrosis are usually small, but sometimes the parenchyma of the liver may be greatly destroyed so as to leave a virtual shell of tissue. The fever is "picket fence" in type; and when jaundice occurs, it is of grave prognostic significance. Anemia is usually present and is often severe. The appearance of multiple liver abscesses secondary to suppurative cholangitis is serious, and in the days prior to antibiotic therapy the mortality was in the vicinity of 95%. Massive antibiotic therapy is now generally recommended.

Massive penicillin therapy has been advised for the treatment of liver abscess,[2] but it has been recognized that free drainage must be obtained or infection will be likely to persist. Weinstein[3] has shown the value of massive doses of intravenous penicillin in the therapy of Gram-negative infections due to the type of organisms ordinarily thought to be penicillin-resistant. They have shown that Aerobacter aerogenes more than any Gram-negative bacillus has a wide range of sensitivity. Only about 38% of the strains were found to be inhibited by 78 units or less per ml. About the same number were susceptible to between 156 and 625 units per milliliter, and of these about 60% failed to grow in the presence of 156 units per ml. Approximately 24% of Aerobacter aerogenes were "resistant." In their series of cases of severe Gram-negative infections treated with massive penicillin therapy, one case of cholangitis due to a mixed culture of B. coli and Aerobacter aerogenes was described who failed to respond to 23 days of therapy. The Aerobacter aerogenes in this case was a resistant organism sensitive only to 10,000 units per ml. It can be said that in our own patient we were fortunate in that the organism in question was sensitive to 500 units per ml., and this was confirmed by the disappearance of the organism from cultures from the bile and from the drain tracts within several days after initiation of massive intravenous penicillin therapy. The reappearance of this particular organism many weeks later was undoubtedly due to the fact that the obstructed and diseased gallbladder is still in situ, but it should be pointed out that reappearance of the organism did not take place until after intravenous penicillin therapy was discontinued.

It is well-known that penicillin is a severe irritant when applied to nervous tissue. Intrathecal administration of penicillin in amounts greater than 50,000 units has frequently resulted in convulsions, pleocytosis and other evidence of central nervous system damage.[11] Although conventional parenteral doses of penicillin do not produce clinical nervous symptoms, electroencephalographic abnormalities have been reported.[12] In Weinstein's published experience with "massive" intravenous doses (20 to 80 million units daily) three instances of convulsions occurred. He believes that generalized convulsions are more likely to occur in people with epileptogenic foci. More usually the central nervous system reactions consist of generalized muscular twitching with exacerbation in response to auditory and tactile stimuli. Penicillin is excreted to the extent of 80% by the tubules and 20% by glomerular filtration.[13] Weinstein therefore recommends reduced doses in old people, who have diminished tubular excretory capacity,

and in infants of one month or less, as well as in the presence of renal failure. In kidney shutdown a single dose of 10 million units is followed by assays of drug levels in the blood. In our patient with evidence of renal tubular damage neuro-irritability finally ceased when the dose was reduced to 2 million units every six hours.

The recent availability of sodium penicillin eliminates the hazard created by the administration of 1.5 milliequivalents of potassium ion with each million unit of the commonly used potassium salt of penicillin-G[3] in patients with renal failure, thus further enhancing the safety of massive penicillin therapy.

Conclusions

A case of suppurative cholangitis due to mixed organisms, treated with "massive" intravenous penicillin therapy in conjunction with cholecystostomy and drainage of multiple liver abscesses is reported.

The cause of the ascending bile-duct infection in this patient is not known, but it is presumed to be secondary to empyema of the gallbladder and a subsequent common-duct obstruction due to inflammatory swelling in the vicinity of the portal triad. No evidence of cholelithiasis was found.

The severity of the diffuse muscular twitching and the frequency of the generalized convulsive seizures observed during the course of massive intravenous penicillin therapy were directly proportional to the dosage of the penicillin administered to this patient with postoperative renal failure.

No harmful sequelae or neurological deficits have been observed in the two months which have elapsed since the discontinuation of intravenous penicillin therapy.

Sodium Penicillin-G which is now available makes the use of massive penicillin therapy feasible in patients with renal failure without fear of hyperkalemia. Dosage should be reduced if signs of neuromuscular irritability occur.

Acknowledgment

The advice of Dr. Louis Weinstein is gratefully acknowledged.

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The Plight of the Fetus

There are certain drugs which are very hazardous to the fetus. There are others which, experiments indicate, may be dangerous. Then there are those drugs which are circumstantially suspected of being hazardous. This leaves few drugs available for use. Medication cannot stop altogether while physicians wait for the future to provide the answers. As current guide lines, when the parturient needs treatment, drugs must be judged not on the impossible basis of absolute safety, but on a basis of no known added risk. With such a standard, the physician can continue to prescribe for his patients. — Editorial in **Massachusetts Physician,** 23:7, (Mar.) 1965.

Latent Hemochromatosis In A Twin

FRANCIS A. SPELLMAN, M.D.*

Controversy continues as to whether hemochromatosis is a disease of genetic origin or is merely acquired by eating and absorbing too much iron. MacDonald[1,2] has stated the thesis that hemochromatosis results from ingesting an excess of iron. Williams, *et al*,[3] studied clinical and liver biopsy material in 46 relatives of 16 patients with idiopathic hemochromatosis. Increased amounts of iron were found in the relatives and did not appear to be of environmental origin. Thus to the latter authors hemochromatosis seems to represent a metabolic defect which may be modified by environmental factors such as alcoholism and diet.

The purpose of this paper is to record the finding of asymptomatic latent hemochromatosis in the twin brother of a patient who died at this Center of the cardiac and hepatic effects of idiopathic hemochromatosis. The diagnosis in the case of the deceased was confirmed by autopsy findings, while that of the asymptomatic twin brother was made by elevated serum iron levels and liver biopsy. Since the brothers had little in common environmentally after childhood, the case presented seems to favor the concept of a genetic defect in certain instances of this disease. That their environments differed radically in the 25 years after adolescence may be important in evaluating the etiology of abnormal iron metabolism in each of these individuals. One prerequisite for the development of hemochromatosis appears to be the presence of enough iron in tissues over a sufficient interval of time (5 to 45 years[4]) to produce hepatic and other tissue damage.

Case History

A 38-year-old accountant was admitted to the Togus Veterans Administration Center on September 3, 1963, to be examined for possible evidence of latent hemochromatosis. His twin brother had died at this Center a few months before of the cardiac and hepatic complications of idiopathic hemochromatosis. The patient felt perfectly well at the time of admission. Five years previously epigastric distress had occurred and his physician found his spleen to be enlarged. A G-I series was done which was negative. On two subsequent physical examinations in the past 3 years no splenomegaly was noted. Palpitation had occurred occasionally as well as mild asthma. The patient had incurred shell fragment wounds of the left elbow and both legs in World War II.

There was no history of jaundice, use of alcohol, or diabetes. His twin brother had traveled widely about the world and had used alcohol to excess over a long period of time. Dietary history for excess iron ingestion was negative.

Physical Examination revealed a blood pressure of 150/80. No abnormal pigmentation was noted. No stigmata of liver disease were present. General physical examination was negative.

Laboratory Data: Hematocrit, 48; hemoglobin, 16 Gm.; white blood count, 6500, 62% neutrophiles, 38% lymphocytes; urinalysis, negative. Liver functions were negative. Glucose tolerance test was normal. Serum iron, 297 gamma/100 ml. and 252 gamma/100 ml. on repeat. Iron binding capacities were not done at this time.

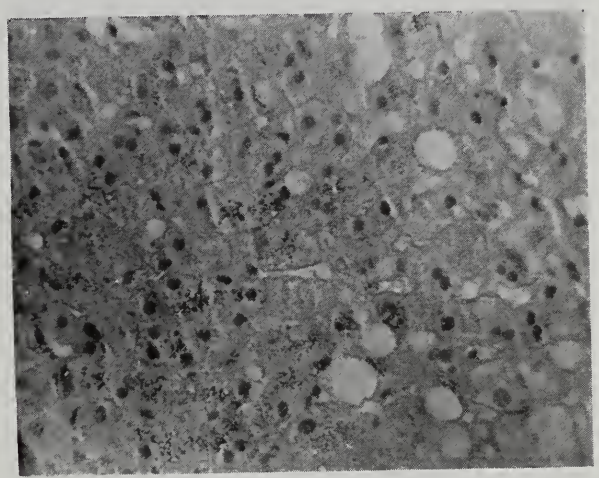


Fig. 1. Liver biopsy, hematoxylin and eosin stain, Sept. 1963. Many liver cells contain a refractal light brown granular pigment.

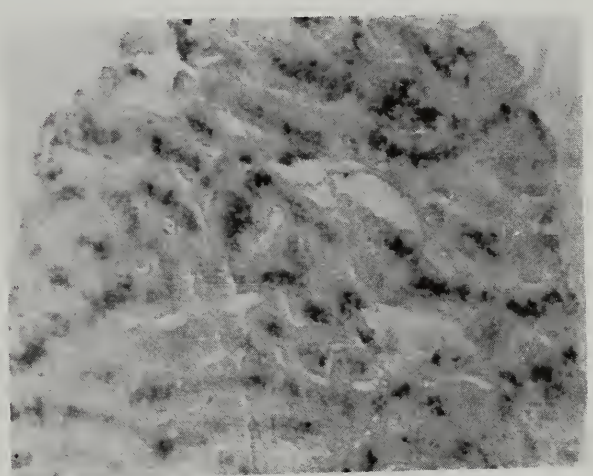


Fig. 2. Liver biopsy, iron stain, Sept. 1963. Positive for iron in liver cells.

Electrocardiogram was normal. Chest x-ray was negative as was a scout film of the abdomen.

Liver biopsy revealed a normal architecture but on iron staining a definitely increased and abnormal amount of iron was noted in most liver cells without evidence of hepatocellular damage. (Figs. 1 and 2)

Since the diagnosis of latent hemochromatosis had been established, a prophylactic program of weekly phlebotomies was initiated on September 25, 1963. The purpose of the program was to remove excess iron stores and thus prevent onset of clinical hemochromatosis.[6] By December 20, 1963, hemoglobin was still 13.9 Gm., hematocrit, 44; serum iron, 132 gamma/100 ml.; iron binding capacity, 368 gamma %. On January 14, 1964, serum iron was 55 gamma % and iron binding capacity 294 gamma %. On March 6, 1964, serum iron

*Chief, Gastroenterology Service, Veterans Administration Center, Togus, Maine.

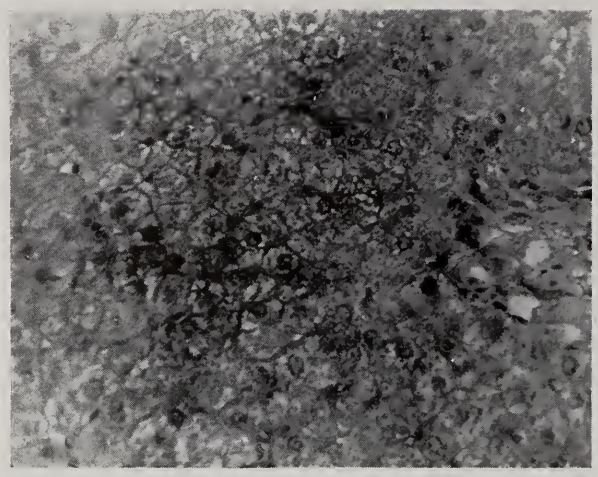


Fig. 3. Liver biopsy, hematoxylin and eosin stain, Sept. 1964. Liver architecture is normal.

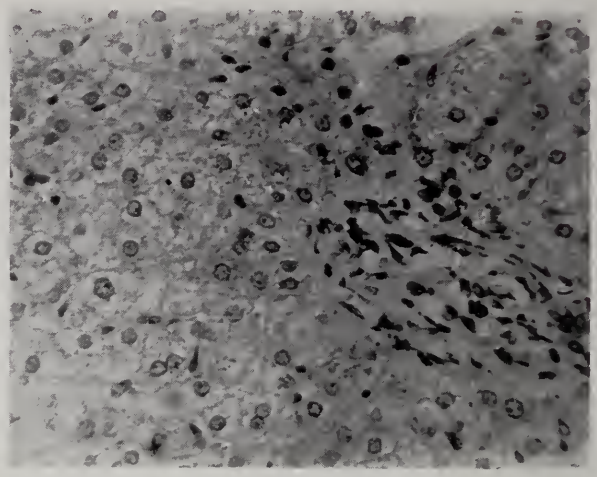


Fig. 4. Liver biopsy, iron stain, Sept. 1964. Amount of iron is considered normal.

was 39 gamma %; hematocrit, 36; hemoglobin, 11.7 Gm. At this point, phlebotomies were extended to monthly intervals.

The patient's second admission was on September 8, 1964, for the purpose of a liver biopsy. He stated that he felt perfectly well except for mild fatigability and discomfort in the right wrist on vigorous use of this joint. Physical examination was again normal. Laboratory data was as follows: Hematocrit, 44; hemoglobin, 13.1 Gm.; uric acid, 4.8 mg.; serum iron, 32 gamma %; iron binding capacity, 383 gamma %; 24-hour uric acid excretion, 540 mg. X-rays of the wrists revealed eburnation of the articular surfaces of the radius, navicular, lunate, and greater and lesser multangular bones bilaterally. These changes raised the possibility that the patient may be developing arthritis of metabolic origin as reported by Schumacher.[7] Liver biopsy showed an amount of iron probably not above what is normally present. (Figs. 3 and 4) Liver architecture was normal. The patient was discharged with advice to have a phlebotomy every 2 months in an effort to prevent reaccumulation of excess liver deposits of iron. On two subsequent follow-up visits hematocrit and hemoglobin have been normal and serum iron was 50 gamma % on January 22, 1965.

Discussion

The clinical syndrome of idiopathic hemochromatosis (bronze diabetes) is accompanied by pigmentation of the skin, hepatomegaly and subsequent liver insufficiency, diabetes mellitus, and eventually one third of all cases die of cardiac failure.[4] Cardiac arrhythmias commonly accompany the congestive failure which is often right-sided in character. Latent hemochromatosis, on the other hand, is a term used to describe abnormal deposition of body iron stores in parenchymal tissues such as the liver, which have not as yet led to tissue damage and fibrosis. An excess of parenterally administered iron, for example from prolonged transfusion therapy, results in deposition of large amounts of iron in the reticuloendothelial system (hemosiderosis). As redistribution of this iron occurs with the passage of time, clinical hemochromatosis may develop from damage to liver, pancreas, heart, and other parenchymal tissues. Enthusiasm for the concept of heredity as the vital etiological factor in idiopathic hemochromatosis must be tempered by consideration of the findings of Pechet and MacDonald[8] that 58% of an unselected autopsy series had stainable iron deposits in the liver, indicating that increased liver iron stores are more common than has been previously documented. We believe however that the case herein reported in a twin lends strength to the importance of heredity in this disease and also emphasizes the important role of liver biopsy in evaluating relatives of patients with hemochromatosis. When increased liver iron stores are found in such relatives, a prophylactic program of venesection should be instituted.[6] Use of chelating agents to reduce abnormal iron stores may be a hopeful technique for the future.

Conclusion

A case of latent hemochromatosis is presented which occurred in the twin brother of a patient who died of the cardiac and hepatic effects of idiopathic hemochromatosis. The relative roles of heredity and environment in the etiology of disorders of iron storage are discussed. Removal of excess liver iron deposits by a program of venesection has been demonstrated by the above study. The importance of searching for latent abnormal iron storage in the relatives of patients with hemochromatosis is stressed.

Acknowledgment

The author wishes to express appreciation to Robert L. Ohler, M.D., Chief, Medical Service, Veterans Administration Center, Togus, for performing serial liver biopsies on this patient and to the blood bank at The Maine Medical Center, Portland, for performing serial phlebotomies.

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Perforation of Stercoraceous Ulceration of the Colon

Kenneth E. Smith, M.D.*

Spontaneous or idiopathic perforation of the colon, excluding perforations associated with carcinoma, diverticulitis, obstruction and trauma, is a rare occurrence. Castleton reviewed 11 reported cases and added one of his own.[1] The perforation occurred in the sigmoid colon in all of these cases, and in each instance the bowel was reported as being normal in appearance, although in several the left colon was packed with hard fecal masses.

Case Report

R. G. a 68-year-old man was readmitted to this hospital because of difficulty in swallowing and head pain. A left hemiglossectomy and mandibulectomy with radical neck dissection had been done elsewhere 4 months previously. A salivary fistula had developed and gradually closed. A tracheotomy tube was in place. There was no evidence of recurrence of the carcinoma, but because of continued dysphagia, a feeding gastrostomy was done. Codeine was required for relief of pain. He failed gradually without obvious cause. Two days prior to death he complained of abdominal pain and signs of peritonitis developed. This was attributed to diverticulitis, and because of his debilitated condition, he was treated conservatively with suction, fluids and antibiotics with initial improvement followed by rapid worsening of his condition.

Postmortem examination disclosed a generalized peritonitis with fecal contents in the peritoneal cavity. There was perforation of the transverse colon at the splenic flexure which measured 5 cms. in the greatest diameter. There were several well demarcated smaller areas of ulceration of the mucosa in the transverse colon. The entire colon was packed with hard fecal masses. Other findings included a small undifferentiated bronchogenic carcinoma with a cerebral metastasis. There was no evidence of recurrence of or metastasis from the previously resected epidermoid carcinoma of the floor of the mouth.

Discussion

Ulcerations of the mucosa of the colon due to pressure from hardened fecal masses have long been described, but only a few instances of perforation of such ulcers have been reported.[2,3]

Grinvalski and Bowerman reported that they found 10 cases of stercoraceous ulcers at autopsy, in 4 of which perforation had occurred.[3] Constipation had been noted in 6, diarrhea in 3 with rectal bleeding in 2 of these. In 1 case history was not obtainable. In 8 of the 10, the ulcerations, either single or multiple, were limited to the rectosigmoid; in 1 case they were localized to the ascending colon, and in 1 they were distributed in the cecum, transverse colon and rectosigmoid.

The ulcers were described as having sharp irregular outlines and ranging from 2.5 cms. to 10 cms. in greatest diameter. There was depression of the ulcerated surface below the mucosal base line with stretching and thinning of the involved intestinal segment. The perforations occurred in the center of the ulcerations where the wall was thinnest.

Sections of the ulcers showed ischemic necrosis of the mucosa. Numerous bacteria were present in the necrotic tissue. A mild inflammatory reaction was present with mononuclear cells and a few polymorphonuclear leukocytes within the wall. In the case in which perforations had occurred, there was a suppurative peritonitis.

Summary

A case of perforation of stercoraceous ulceration of the transverse colon is reported.

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*Chief, Laboratory Service, Veterans Administration Center, Togus, Maine.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

New Cancer Control Service

Dr. Stanley Beckerman, Director of the Division of Cancer Control, has recently returned to the State of Maine. He has been away since October, 1964, at which time he embarked upon a Research Fellowship at Memorial-Sloan-Kettering Center for Cancer and Allied Diseases in New York City. One of the largest of its kind in the world, the Memorial-Sloan-Kettering Center is devoted exclusively to the care and management of patients with malignant disease. Cooperative studies are carried on between this institution and other institutions around the world, including the Cornell Medical School (New York Hospital) and The Rockefeller Institute, both of which are just across the street from Memorial Center.

Dr. Beckerman's activities centered around the Department of Cancer Chemotherapy, which is under the chairmanship of Dr. David Karnovsky. During his fellowship, he had the opportunity of working with many experimental cancer chemotherapeutic agents which were still in research. Much time was also spent in the Division of Applied Chemotherapy, where anti-cancer agents were employed which had been released for distribution and approved by the Food and Drug Administration.

In addition to becoming proficient in the use of anti-cancer drugs, Dr. Beckerman also worked with many of the specialty groups, such as the Lymphoma Service, the Gastric and Mixed Tumor Service, the Breast Tumor Service, etc., gathering information on the newer techniques in the diagnosis and treatment of malignant diseases.

While in New York, Dr. Beckerman visited the Strang Clinic, a world famous cancer detection center. At this clinic, under the direction of Dr. Emerson Day, seventy-five to ninety patients are seen daily, five days a week, for complete cancer detection physical examinations. Every female receives a cervical Pap smear and, where indicated, bronchial cough specimens are obtained for Pap smears.

Several weeks were also spent in Boston at the Children's Cancer Research Foundation, also known as The Jimmy Fund Building. This institution, headed by Dr. Sidney Farber, is one of the largest of its kind, devoted entirely to the care of children with malignant disease. Techniques were observed in the handling of patients with childhood leukemia as well as children with solid tumors.

The purpose of this program was to enable Dr. Beckerman to bring back to the State of Maine many of the newer concepts regarding the diagnosis and management of malignant disease. New relationships and associations have been established with these various centers outside the State. The Division of Cancer Control now stands ready to assist Maine physicians in an advisory capacity whenever they desire. This may be carried out by telephone or by mail. While this should not be construed as an offer of free direct, continuing service to individual patients, it is a general consultation service, intended to help raise the level of care of cancer patients. It is hoped that advice and suggestions may be made which will prove to be helpful in problem diagnoses of malignant disease. In addition, assistance is being offered in planning the care and long-term management of the cancer patient. Decisions regarding the particular type of therapy to be used at any point in the course of malignant disease is often critical. Frequently, the physician may find it beneficial to seek this type of advisory service before such decisions are finalized. Obviously, the Division of Cancer Control will not have a great many of the answers which are sought. However, through proper channels and contacts beyond the State of Maine, authoritative replies should be forthcoming.

While Dr. Beckerman will continue to direct the Cancer Control Program, he will return to a part-time arrangement, since he will again re-open medical offices in Waterville. His practice will be devoted entirely to medical oncology, the medical management of malignant disease.

Continued on Page 190

Elected at the 112th Annual Session of the Maine Medical Association

Rockland, Maine June 13, 14, 15, 1965

President-Elect
GEORGE E. SULLIVAN, M.D., Fairfield


DR. SULLIVAN

Councilor, Third District
EDWARD K. MORSE, M.D., Rockland

Councilor, Fourth District
JOHN D. DENISON, M.D., Gardiner

Council Chairman
PAUL S. HILL, JR., M.D., Saco


DR. MORSE


DR. HILL

Maine Heart Association Notes

116 State Street, Augusta, Maine

The Supraclavicular Arterial Bruit

"- - - bruits or murmurs arising outside the heart may be transmitted to the precordium and thus be misinterpreted as being of cardiac origin. This problem has been described with regard to the cervical venous hum. During the past 6 years, we have examined a number of children and young adults in whom an arterial systolic bruit, although loudest in the supraclavicular fossa and over the lower common carotid artery, was heard at the base of the heart and was originally thought to arise from aortic or pulmonary stenosis. Because its exact site of origin is unknown, - - - we have elected to call this murmur "the supraclavicular arterial bruit." - - -

"It appeared likely that this benign supraclavicular arterial bruit could be confused - - - with the murmurs of congenital or rheumatic valvular heart disease in children, but also with the bruits of acquired arteriosclerotic disease of the carotid and subclavian arteries in adults. - - -

"The supraclavicular arterial bruit is a common normal finding, especially so in children and in young adults. - - -

"The mechanism of production of this bruit is unknown. - - -

"The supraclavicular bruit is of short duraction - - -, In most instances, it occupies less than half of systole - - -.

"The supraclavicular bruit may suggest aortic or pulmonary stenosis when referred to the base of the heart. Its delayed onset, its brief duration, and its greater intensity in the neck, and the effect on it of compression of the subclavian artery will make the distinction clear.

"The supraclavicular bruit may be distinguished from the bruits of carotid artery disease and organic obstruction of the subclavian artery by its relatively brief duration, and, as a rule, by its occurrence in children and younger adults without a history suggestive of impaired cerebral blood flow."

Submitted by Jacob B. Dana, M.D.

Reference: Fowler, N. O., and Marshall, W. J. American Heart Journal, Volume 69, pages 410-418, 1965.

Recommended Dosage of Lomotil Liquid to Control Diarrhea

File: Diarrhea

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Age	INITIAL LOMOTIL LIQUID DOSAGE*
3-6 mo.	½ tsp. t.i.d. (3 mg.)
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1-2 yr.	½ tsp. 5 times daily (5 mg.)
2-5 yr.	1 tsp. t.i.d. (6 mg.)
5-8 yr.	1 tsp. q.i.d. (8 mg.)
8-12 yr.	1 tsp. 5 times daily (10 mg.)
Adult	2 tsp. 5 times daily (20 mg.) (or 2 tablets q.i.d.)

*Based on 4 cc. per average teaspoonful.
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Cautions and Side Effects
Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

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SEARLE *Research in the Service of Medicine*

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Viruses and Blood Vessel Repair

PHILADELPHIA, PA. — The American Cancer Society announced on July 15, 1965 that scientists at Hahnemann Medical College have found that a mouse leukemia virus with startling promptitude knocks out a major blood vessel repair system.

The findings may offer a partial explanation for the frequent hemorrhage in leukemic patients.

The scientists are Dr. Lewis Hilliard Dennis, a clinical fellow of the Cancer Society, and Dr. Isadore Brodsky, head of the college's Hematology Section.

In these experiments, the investigators used the Friend virus which kills 80% of mice within three months after injection. A few of the mice die of hemorrhage of the spleen, which often ruptures 10 to 30 days after infection; the others die of leukemia 50-80 days after infection.

The Hahnemann scientists found that a short two days after infection the viruses had begun to attack the vessel-repair system.

In mice and in men, ruptured blood vessels normally are patched up with sticky little blood discs called platelets. Platelets are produced in profusion by large blood cells called megakaryocytes, which originate in the spleen.

The targets of the leukemia viruses were platelets and megakaryocytes, the platelet factories. The viruses wasted little time getting to their targets.

Within 48 hours after infection with the Friend virus, the number of platelets in the mouse blood had decreased by one-fourth. As the disease advanced the platelet count fell progressively; it dropped much more rapidly in hemorrhagic disease than in leukemia. A day before death, the count was down to less than one-tenth normal.

Dr. Brodsky and Dr. H. B. Levy showed, with radioactive phosphorus, that following infection the metabolic activity of the spleen cells speeds up a great deal. The splenic cytoplasm becomes loaded with RNA, or ribonucleic acid. The spleen enlarges palpably a week or so after infection, and at time of death it usually is more than three times its normal size.

Cell protein factories are composed of RNA. The active material in a Friend virus also is RNA.

The Hahnemann research indicates that this sequence of events takes place when a Friend virus infects a mouse: 1) the virus invades megakaryocytes in the mouse spleen; 2) virus RNA takes the place of normal cell RNA in tiny cell particles; 3) the RNA in infected cells now produces virus instead of the normal products, particularly platelets; 4) the spleen grows rapidly because of a neoplastic growth of reticulum cells that are also attacked by the virus; and 5) with the complement of platelets depleted and the vessel vulcanizing system crippled, the animal bleeds to death as small blood vessels rupture.

While a growing number of animal leukemias now are known to be caused by viruses, evidence of virus involvement in human leukemia is still highly circumstantial and sketchy. Nevertheless, should a virus come to be involved, the Hahnemann studies well may suggest their mode of action in human disease.

From American Cancer Society News Service Release

Announcements

New England Society of Anesthesiologists

The Annual Fall Conference of the New England Society of Anesthesiologists will be held on September 16, 17 and 18, 1965 at Wentworth-By-The-Sea, Portsmouth, New Hampshire. The guest faculty will include: Drs. Nicholas Greene, Solomon Hershey, William Derrick, John Steinhaus and James Eckenhoff.

American College of Nutrition

The annual scientific meeting of the American College of Nutrition will be held at the Americana Hotel in New York City on Sunday, October 10, 1965, at 10 a.m. The topic will be "Nutrition — Alcohol — Office Practice." For further information, please contact Robert A. Peterman, M.D., F.A.C.N., Secretary, 3 Craig Court, Totowa Borough, New Jersey, 07512.

Book Review

Brain-Thyroid Relationships with Special Reference to Thyroid Disorders. Edited by Margaret P. Cameron, M.A., and Maeve O'Connor, B.A. Ciba Foundation Study Group No. 18. Little, Brown and Company, Boston, 1964, 117 pp., $1.95.

Brain-thyroid relationships are discussed by a group of investigators from England, United States, Scandinavia, and Turkey in a very bright little book that holds an evening's worth of pleasure. The initial chapters deal with the interaction of the hypothalamus, pituitary, thyroid. The role of TSH, thyrotropic releasing factor, and vasopressin are fully discussed. There is an excellent description of the anatomy including electron-microscopy of the median eminence of the hypothalamus and its possible role in the release of thyrotropic-releasing factor. According to most recent concepts the anterior pituitary releases TSH which acts on the thyroid to release thyroxin. The latter inhibits further TSH secretion if the level of thyroxin becomes excessive. The hypothalamus releases thyrotropic releasing factor which stimulates the anterior pituitary to release TSH. The hypothalamus is temperature and calorie sensitive.

The importance of the thyroid in cerebral development, I.Q. studies in congenital hypothyroidism, and psychological factors in hyperthyroidism constitute the final chapters.

Each of the chapters is self-contained and includes a vigorous discussion. The book is very compact, well bound, and contains a few well selected photographs and graphs. The aim of the book, to discuss brain-thyroid relationships is well accomplished in 113 pages. The book does not go into the full details of thyroid metabolism. The chief emphasis is on the brain aspects. This monograph is quite useful for the clinician, physiologist, and medical student.

PETER F. LANSING, M.D.
Augusta, Maine

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DEPARTMENT OF HEALTH AND WELFARE

Continued from Page 184

Miss Gay Retires

After thirty years of service with the Department, Katharine D. Gay, Administrative Assistant, Division of Tuberculosis Control, retired on July 3, 1965. A native of Damariscotta, Maine, Miss Gay attended the schools of that community and was graduated from Lincoln Academy in 1922. She then entered Northfield Academy in Northfield, Massachusetts and was graduated in 1924. The following autumn, she entered Simmons College in a 5-year baccalaureate nursing training course, receiving her B.S. from the College in 1929 and an R.N. from the Massachusetts General Hospital at the same time. In 1930, she received post-graduate training at Boston University in the area of health education; the two years following, she served as school nurse in the Stoughton, Massachusetts and Hackensack, New Jersey public schools, respectively. In 1933, she was Acting Superintendent of the Boston Nursery for Blind Babies.

Nineteen hundred and thirty-four saw Miss Gay in Maine again, this time as staff nurse for the out-patient department of the Franklin County Memorial Hospital, Farmington. The following year, she began her work in the field of tuberculosis, serving as clinic nurse for the Maine Public Health Association (predecessor of the Maine Tuberculosis and Health Association.) In 1935, she entered the service of the State as a member of the staff of the Division of Tuberculosis Control.

Long a leader in the field of tuberculosis control — in administrative capacity — Miss Gay has forged steadily ahead, keeping abreast with every development in thinking and action in this direction. Of inestimable help always to her fellow workers in the Department, to the allied health agencies and groups in the field and to the private physician in his office and the hospital, Miss Gay has served with both strength and humility. Her thought was always "for the patient."

Over the years, she has received many honors attesting to the regard in which she was held throughout this State and abroad. She will be missed.

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Contraindications: Hypersensitivity and most cases of severe renal or hepatic disease.
Precautions: Reduce dosage of concomitant antihypertensive agents by at least one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take special care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended.
Side Effects: Agranulocytosis, constipation, dizziness, dysuria, headache, hyperglycemia, hyperuricemia, impotence, leukopenia, muscle cramps, nausea, postural hypotension, purpura, thrombocytopenia, transient myopia, urticaria, vomiting and weakness.
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For full details, see the complete prescribing information.

*Swartz, C., et al.: Circulation 28:1042, 1963.

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The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, September, 1965 | No. 9

The "Jumpers" of Maine

Past History and Present Status

E. Charles Kunkle, M.D.*

A curious reaction was noted in Maine woodsmen 87 years ago. Beard in 1878 at an annual meeting of the American Neurological Association briefly described the disorder as found among men in the Moosehead Lake region, featuring an excessive and dramatic startle response to sudden stimuli.[1] It appeared in part to be hereditary. Except for occasional references in foreign literature in the next decade, the disorder apparently quickly dropped out of formal medical notice until an example was reported by Stevens in 1964.[2] A search for other traces, carried out in the past year, yields evidence that this colorful reaction remains endemic in the State of Maine, at least. Its neurologic and psychologic aspects are complex and intriguing.

Early History

The observations which Beard later recorded in moderate detail were based on a trip from New York to Moosehead Lake to interview several typical "jumpers."[3] These men were easily startled by an unexpected sound or touch, sometimes reacting simply by jerking of the shoulders and arms, or, under a strong stimulus, making a more violent motion and crying out as if in alarm. In addition, some would automatically repeat a brief phase addressed to them (echolalia) and would at once obey a forceful command (automatic obedience). Thus, they might jump several inches from the ground, drop or throw an object they were holding, or strike out at a nearby friend, if suddenly ordered to do so. In such apparently involuntary obedience, some jumpers had been known to injure themselves by an inappropriate act, as thrusting a fist through a window or against a hot stove. Because of these propensities, the jumpers were often harassed by their colleagues, who found amusement in provoking them by various means.

Hereditary features were noted in some victims. In one family the father, two grown sons and two small grandchildren, ages 4 and 7, were afflicted. Fourteen cases were identified in four families; almost all were males.

Within the next few years attempts were made by various physicians to find similar disorders elsewhere in the world. Two are particularly noteworthy. Hammond, a retired Surgeon-General of the U. S. Army, described in 1884 a disorder found in Siberia and known to the Russians as Miryachit.[4] It differed from "jumping", however, in featuring automatic imitation of an act suddenly performed by another individual (echopraxia), in addition to echolalia. Actual "jumping" was apparently not characteristic and women were more susceptible than men. For more than a century, another strange disorder has been known in the Malay Archipelago, much resembling the Siberian disorder in showing echolalia and echopraxia; it also includes automatic obedience and is most common in women past the prime of life.[5] As Yap, a physician in Hong Kong, has indicated, both of these exotic disorders, occurring in primitive peoples, seem to represent an unusual state of exaggerated suggestibility. Neither has close resemblance to "jumping".

Except for a passing comment on the "jumpers of Maine" by Osler,[7] who viewed this reaction as an elaborate habit spasm or tic, the American disorder attracted little or no further notice until Stevens described to another annual meeting of the American Neurological Association in June 1964 his case found in Washington, D. C.[2] The patient was a 59-year-old French-Canadian who had been raised in northern Maine and whose excessive startling dated from childhood. The routine neurologic examination was normal and several elec-

*Associate Neurologist, Maine Medical Center, Portland, Maine

troencephalograms were unremarkable, even during a startle response.

Old and Recent Clues

In non-medical circles in Maine, however, "jumping" has had continuing recognition, in both the remote and recent past. The phenomenon is apparently known to many long-term residents of this state and has become part of regional folklore. Holman Day in his "Pine Tree Ballads," published in 1916, describes in "The Jumper" a typical example in a man whose habit dated from a sudden severe fright in childhood.[8] This man jumped so readily and violently while abed when the nightly train whistled as it passed that his wife was repeatedly struck, receiving a black eye "almost 16 times," and finally had to sleep in another room.

The current search for additional leads to the disorder in southern Maine indicates that "jumpers" are far from rare in several coastal cities, including Bath, Brunswick and Portland. All thus far described are male factory employees or other semiskilled workers. The personnel director of one large paper mill identifies 15 instances among his labor group. Various national backgrounds are represented, including French-Canadian, Irish and Italian. One notorious "jumper" in the Millinocket area is reported to be remarkably susceptible to a sonic boom. When startled by this he has been known to heave a sledge hammer as far as 50 feet.

Five typical "jumpers", all male, have been examined by the author in detail. Four of these described a major complication of the disorder in themselves and in a few other similarly afflicted individuals whom they knew. When startled they may quite involuntarily strike out against any person close by, in a coordinated and aggressive manner. The response is sometimes only a threatening gesture, but on a few occasions has included an actual blow or a firm push, leading to actual injury to the innocent bystander. These potentially dangerous "jumpers" have long been tense, restless, and dissatisfied, but none has been particularly belligerent or quarrelsome. They consider themselves to be quite ticklish. One is remarkably intolerant of being touched on his torso, even with adequate warning. He received a medical discharge from the Army, in part, at least, because of his affliction. On the target range, when nudged by the officer in charge as a signal to shoot, he once turned and quickly fired wide of the mark, and was thereafter barred from the range. Another, when startled while dancing with his wife, struck her so forcefully as to knock her to the floor.

Secondary "Jumping"

The phenomenon of "jumping" has medical interest and significance well beyond its occurrence simply in otherwise healthy "jumpers". An excessive startle response is found in some children with major forms of brain injury dating from birth or the neonatal period,[9] but whether this response persists into adult life in these patients is uncertain. The startle response is also enhanced in association with magnesium deficiency.[10] It may develop as a sequel to various illnesses which evoke considerable tension, often with irritability and sometimes with depression. It has recently been observed by the author in a man of 58 following a minor cerebral infarction and in a woman of 66 in association with muscle-contraction headache in a setting of mounting resentment and despondency.

On the other hand, for unknown reasons startling tends to be reduced in intensity in patients with various epileptic disorders.[11] Jumping has only a remote relationship, if any, to "startle myoclonus" found in degenerative encephalopathies[12] or to audiogenic seizures or other forms of "reflex epilepsy."[13]

Discussion

The neurologic and psychologic aspects of "jumping" remain incompletely defined. Excessive startling in various degrees is clearly a common symptom, noted to varying degrees in many individuals, both sick and well, and sometimes occurring only temporarily. In the "jumpers" as found in Maine, both past and present, the reaction may have strong cultural factors. Dr. Reuben Rabinovitch of Montreal, from his observations in a childhood spent in rural southern Canada, has suggested that in some of his countrymen "jumping" dates from a game commonly played by children in which a chosen victim was startled by a noise or a poke.[14] The victim would be expected to produce a rather formalized response in which he would jump violently, flail out and shout angrily. often imitating the cry of a kicking horse. This variety of "horse play" was sometimes carried on into adult life, particularly in isolated villages or in camps of lumberjacks. Among individuals with this heritage, therefore, "jumping" may be represent a particular variety of socially conditioned reflex, even though its origins might be difficult to trace in individual instances. Thus, in some communities it may be part of what Kinnier Wilson has termed a "collective psychoneurosis."[15]

There is much to suggest that the "jumper" may be acting out or otherwise releasing considerable concealed or overt hostility, especially when his reaction includes an aggressive gesture or blow. Recent studies of echolalia and echopraxia are pertinent to this hypothesis, for, as reported by Carluccio et al, "echo" reactions sometimes seem to represent concealed hostility expressed in a unique manner of imitation.[16]

Summary and Conclusions

In short, "jumping" is found in varied forms and degrees in health and illness. In many subjects it is merely a minor quirk, a benign oddity of behavior, a slight exaggeration of a normal response, and does not achieve sufficient status to be a disease. In some, however, it is a striking and lifelong reaction pattern, sometimes representing an embarrassing or dangerous handicap. In some communities, at least in the past, it seems to

have become culturally standardized. It is probably a widespread phenomenon; its apparent predilection for Maine and nearby areas of southern Canada may largely be an artefact of observation.

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Trans-Coccygeal Prostatectomy*

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Although comparatively new in the country, the technique of trans-coccygeal prostatectomy was carried out in Vienna for a number of years prior to 1948 by Hochinegg and in France by Fabre and Draget. In 1954, Thierman in Germany extensively used this approach for prostatectomy. Dr. Colston at Johns Hopkins brought back these reports from the 10th International Congress of Urology in Athens, Greece in 1955.

Dr. Brice Vallett[1] of Wilmington, Delaware, in 1959, reported his impressions on 7 parasacral prostatectomies as this procedure is also termed. This seems to be the first report of such cases in the country. Dr. Hassein Galji[2] of Elkins, West Virginia, reported 5 trans-coccygeal prostatectomies, three conservative and two radical operations. It is interesting to note that some recent unrological texts have not described this procedure.

In becoming more familiar with this operation, it was noted that excellent mobility and exposure of the rectum can be obtained. The general surgeon, therefore, should find this a very good approach for selected rectal lesions. It should be remembered that the Kraske operation for carcinoma of the rectum utilizes this approach. Kraske developed this operation in 1885.

Two reports in the literature are of particular interest to general surgeons. The first is by Dr. Irving Black[3] of the Department of Surgery, State University of New York College of Medicine at Brooklyn, titled A More Radical Perineal Phase for Abdomino-perineal Resection of the Rectum for Carcinoma. In this report he advocates the removal of the coccyx in this operation as did Miles in 1908 as reported in the Lancet.

The second report by Dr. T. F. Moran,[4] Professor of Proctologic Surgery at Hahnemann Hospital, in a report titled *Trans-sacral Removal of Rectal Lesions,* presented an interesting series of 26 cases. In this report Moran points out that this direct approach to rectal lesions located above the sphincters may enable the surgeon to avoid resorting to unnecessary radical procedures. He reported 3 cases of rectal strictures, 15 cases of carcinoma of the rectum (9 malignant adenomas, 6 inoperable cases because of pain and bleeding previously treated by radiation), 6 cases of Villous tumor, 1 case of rectal procidentia, and 1 case of foreign body.

Description of Operation

In performing the operation the patient is catheterized with a #20 Foley catheter with a 5 cc. balloon which is taped in place and left uninflated until completion of the operation. The patient is placed on his abdomen with the hips slightly flexed. The buttocks are separated by means of adhesive on each side. The incision is made in the midline over the lower portion of the sacrum and coccyx and carried around the left side of the anus. The

*Presented at the New England Surgical Society, 1963

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coccyx is readily removed at its junction with the sacrum. The rectum is then easily mobilized by bluntly dissecting it from the areolar tissue supporting it, then sweeping it to the patient's right side where it may be held out of the way with retractors. This allows for excellent exposure of the seminal vesicles and the posterior aspect of the prostate. The urethra is then exposed, a rubber tissue drain is then placed beneath it which allows traction to be used, the urethra cut across just distal to the apex of the prostate, the uninflated Foley catheter can then be slipped out of the bladder, held aside until the prostate and seminal vesicles are dissected off the bladder. After this has been done, the catheter is then replaced to the bladder and the anastomosis of the urethra to the vesicle neck is completed with interrupted 00 chromic catgut sutures. A rubber tissue drain is placed near the site of this anastomosis and brought out through the lower angle of the incision to be removed in two to three days. The rectum is then allowed to fall back into its normal position and the incision closed with chromic catgut sutures.

Results

Sixteen radical prostatectomies for cancer of the prostate using the transcoccygeal approach were done at the Maine Medical Center. One patient had a previous abdomino-perineal resection of the rectum because of cancer of the rectum. One patient had severe arthritis of the hips which made this an ideal approach because it was impossible to place this patient in the lithotomy position. There were no deaths. The length of hospital stay varied from 7 - 14 days. Discomfort in the operative area was present in all the patients, but was not severe and did not last longer than 1 month. One patient did considerable postoperative bleeding, and it was necessary to do a cystotomy. Two patients have slight urinary incontinence, and two patients have strictures which are dilated at intervals of 6 months.

Summary

The technique of transcoccygeal prostatectomy is comparatively new in this country and is not described as yet in some recently published textbooks of Urology.

This operation was used in 16 patients at the Maine Medical Center. It was used only in patients with cancer of the prostate, the radical operation being carried out in all 16 cases.

The transcoccygeal approach gives an unusually direct and excellent exposure of the seminal vesicles and makes their dissection from the bladder easier.

This operation is particularly useful for prostatic patients who are unable to be placed in the lithotomy position because of severe arthritis or other pathology of the hips.

This operation is recommended in patients who have had previous radical rectal or perineal surgery.

With the above experience for comparison, the author still prefers the conventional perineal approach for prostatic biopsies and radical prostatectomies.

This approach for selected rectal lesions should be kept in mind by general surgeons.

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Hemorrhagic Necrosis of the Bowel Without Demonstrable Vascular Occlusion*

Robert P. Andrews, M.D.** and Joseph E. Porter, M.D.***

Introduction

In recent years, we have noted a number of cases of necrosis or partial gangrene of the bowel in which vascular occlusion of major vessels could not be demonstrated. The pathogenesis of this entity as described by Drucker et al and called hemorrhagic necrosis[1] has not been clearly defined. As a result of a representative case discovered here at autopsy early in 1965, we reviewed our records for the past ten years to search for a possible common pathophysiologic mechanism.

Case Material

In the past ten years there have been 31 cases of hemorrhagic necrosis of the bowel documented at the Maine Medical Center, either at surgery or autopsy or both. Of these, nine have been selected for study because there were no demonstrable ante-mortem vascular thromboses in the major vessels.

The ages of these nine patients ranged from 58 to 84 and there were four males and five females included, whose average age was 70. Their clinical course varied in length from the shortest which lasted 16 hours after the onset of symptoms until death, to the longest which was symptomatic for two years until a terminal exacerbation lasting three days occurred.

The commonest symptoms were abdominal pain in six and vomiting in five, but nausea, diarrhea, and gastrointestinal bleeding were variably present as well. All the patients had objective evidence of gastrointestinal hemorrhage, either as guaiac positive stool, coffee-ground vomitus, or gross bleeding. Surgical exploration was carried out in four and in two, resection was attempted. One of these patients died at surgery and the other proved un-resectable because of diffuse involvement.

Digitalis had been used in seven of the patients for variable periods but the presence of toxicity was established in only one. Adrenalin was utilized during resuscitative attempts in three without lasting improvement.

Four of the patients had a history of hypertension and six had a history of atherosclerosis as revealed by electrocardiographic abnormalities such as evidence of previous myocardial infarction or atrial ventricular block. The terminal series of events in seven of the patients were those of predominantly central circulatory failure as described by Harrison[2] and Sodeman.[6] Respiratory rales, venous engorgement, cardiac arrhythmia, edema, and cyanosis were present, all of which suggest an inadequate response of the heart to the inflow circulatory load. One patient exhibited both central and peripheral circulatory failure. Another had predominantly peripheral circulatory failure with sudden collapse, hypotensive shock, pallor, and tachycardia after a protracted three-day course of gastrointestinal bleeding.

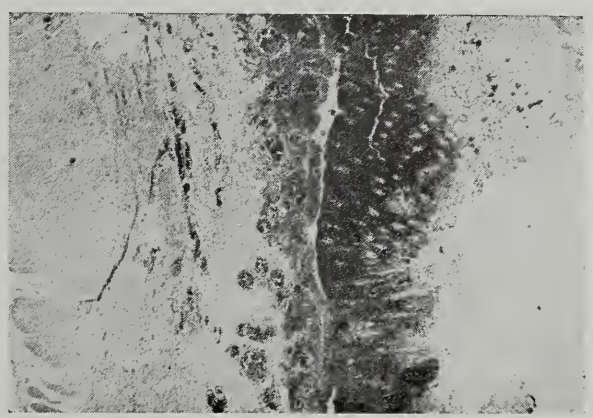


Fig. 1. Mucosal hemorrhage and superficial necrosis with congestion of the underlying vessels. X35

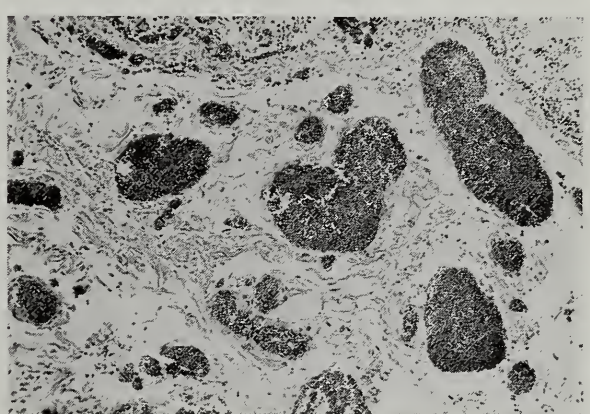


Fig. 2. A striking degree of venous congestion is apparent in these submucosal vessels. X110

Post Mortem Findings

At autopsy, all nine cases exhibited the characteristic gross hemorrhagic appearance of infarction.[4] Small intestine involvement was present in all cases, and in seven the process involved part or all of the large intestine as well. The distribution of the lesions did not

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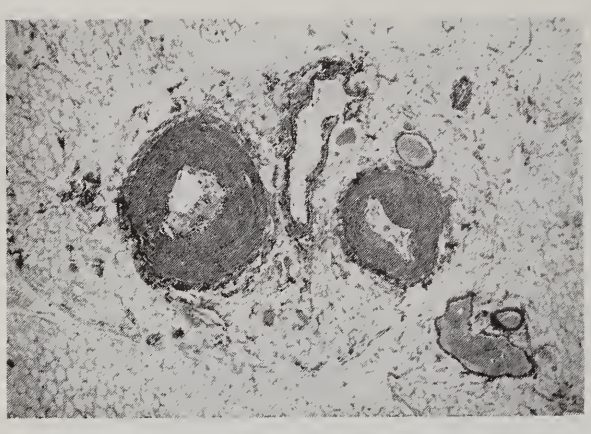


FIG. 3. Marked narrowing of small arterial lumina due to intimal thickening and medial hypertrophy. X65

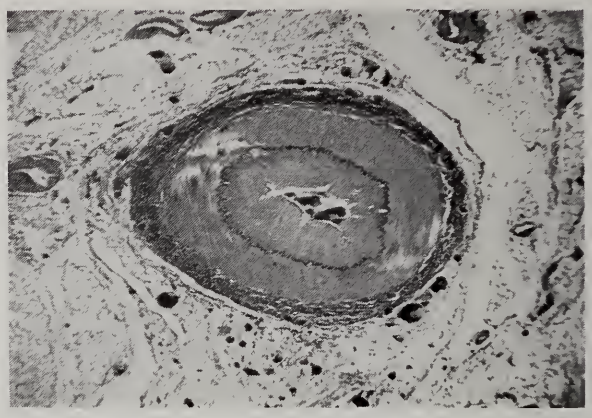


FIG. 4. Marked intimal thickening with a normal elastica interna (Verhoeff's Stain). X65

in any consistent way correspond to the anatomical vascular pattern. The typical gross appearance was that of dusky or mottled cyanosis, with dilatation and thinning of the bowel wall, and hemorrhagic involvement of the mesentery. The bowel lumina contained grossly bloody material in all the cases described. Careful dissection of the mesenteric vessels failed to reveal either thrombotic or atherosclerotic occlusion. It is notable that at autopsy eight of nine patients had a demonstrable degree of atherosclerosis in the aorta or elsewhere which did not affect the mesenteric vessels.

The microscopic appearance varied in severity. The degree of necrosis and edema present in the intestinal wall is listed in Table 1.

The presence of intimal or medial thickening of the mesenteric vessels, venous congestion, and vasculitis was also ascertained and is listed in Table 2.

Other findings of interest included recent venous thrombi in two cases which were interpreted as agonal in origin. In one case in which surgical exploration of the mesenteric artery took place, there was a dissecting hemorrhage of this vessel. This was attributed by the attending surgeon and the pathologist to operative manipulation. Special stains revealed no consistent elastic tissue change in the mesenteric vessels but seven patients had a significant degree of medial or intimal thickening. The microscopic sections of the liver revealed a concomitant degree of central lobular congestion in each case, which had progressed to necrosis of the adjoining parenchymal cells in some instances.

TABLE 1

Microscopic finding	Present (in)
Mucosal necrosis	9 or 100%
Submucosal necrosis	6 or 67%
Muscular wall necrosis	2 or 22%
Interstitial edema	2 or 22%

TABLE 2

Microscopic finding	Present (in)
Thickening of intima	4 or 44%
Thickening of media	7 or 78%
Diffuse venous congestion	9 or 100%
Established vasculitis	3 or 33%

DISCUSSION

The presence of hemorrhagic necrosis of the intestine following an arterial or venous infarction due to thrombosis is well documented.[4] The presence of hemorrhagic necrosis without demonstrable occlusion has been noted by many investigators and led at least one pathology resident to insure the surgeons presence prior to dissection in order to prove the point.[5] Yet others still assert that "in practically all cases of bowel infarction, some kind of vascular obstruction or narrowing can be found in the mesentery or intestinal wall if looked for."[16] It is possible that this difference of opinion, which implies casual examination on the part of investigators unable to demonstrate occlusion, arises from a difference in interpretation of the intestinal micro-pathology. The differences apparent histologically embrace three entities which have a similar gross appearance. Table 3 lists the histologic differences.[1] In addition, as noted above, the lesions of hemorrhagic necrosis were unrelated to the vascular pattern.

The effects of vascular spasm have been implicated by Gazes et al who describe its location in the liver sinusoids associated with Digitalis use and intoxication.[9] Spasm in the hepatic vein,[9] and in the arterial supply of the bowel[5,7,8,10,11] have also been incriminated. There is no question that spasm can occur following the use of vasoconstrictors[12] and may occur as a complex response to hydrocortisone, bacterial endotoxins, or immunologic phenomena. Indeed, though hypotension alone in dogs can result in intestinal vascular spasm and hemorrhagic necrosis,[10] the location of the spasm and resultant degree of splanchnic pooling is disputed. One investigator suggests that the significant redistribution of blood which does occur favors the splanchnic areas[13] while another suggests that in the same situation the splanchnic circulation shuts down altogether, and any increase in portal pressure is a result of hepatic vaso-spasm.[10] Of unknown significance is the anatomic difference in the

Table 3

INFARCTION
Vascular thrombosis
Diffuse edema
Bacterial colonization (variable)
Necrosis and perforation
Segmental distribution
PSEUDOMEMBRANEOUS ENTEROCOLITIS
Mucosal denudation
Marked leukocytic infiltrate
Fibrin and desquamated cells forming a pseudomembrane
Late lymphoid ulceration
HEMORRHAGIC NECROSIS
Mucosal necrosis
Minimal edema
Marked submucosal hemorrhage
Diffuse venous congestion

intestinal blood supply of dogs and man. In dogs the smaller arteries split to each supply one half of the bowel circumference, whereas in humans, each segment is supplied by alternate vessels.[10]

Aortic insufficiency[14] and atherosclerosis have been implicated in the past, but we could not demonstrate either lesion in our cases. Intimal thickening of small arteries has been implicated in another study,[15] and in all of our cases there was some degree of microscopic narrowing of the smaller arteries due to either intimal or medial thickening or both.

These nine elderly patients all demonstrated three consistent anatomic pathophysiologic findings: central circulatory failure, diffuse venous engorgement, and progressive superficial necrosis of the bowel. The presence of a reduced cardiac output with a prolonged reduction of mesenteric and systemic flow and impaired oxygenation of the intestine could produce the pathologic findings. The mucosa is most severely affected, a phenomenon most likely due to heightened sensitivity to hypoxia and the action of the intestinal flora. Further study is necessary to establish the latter hypothesis. If there is added any degree of spasm or vascular narrowing such as was found in the smaller arteries, a working hypothesis may be formulated. Our findings support the conclusions of Champton,[8] Ende,[16] and Drucker, who performed independent studies of the same entity. The most likely mechanism is a combination of factors that taken together cause a profound hemodynamic dysfunction in the splanchnic vascular complex. This results in progressive hemorrhagic necrosis of a portion of the intestinal wall, and the clinicopathologic syndrome of mesenteric infarction.

Summary

A series of nine cases in the past ten years in which hemorrhagic necrosis of the intestine occurred without demonstrable vascular occlusion has been presented. The presence of certain pathologic findings in these elderly people which were common to all cases led to the postulation that a prolonged reduction in cardiac output and small artery insufficiency are the prime factors in a combination of alterations which cause a syndrome clinically indistinguishable from mesenteric infarction.

Acknowledgment

We would like to express our appreciation to Mr. James MacKenzie for the photographs.

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Adaptation of the Self Thromboplastin-Prothrombin Time to an Automatic Prothrombin-Timer and its Reproducibility*

Ugur Derman, M.D.**

The ST-P time is a clotting test which has been proposed recently for the screening of hemorrhagic diathesis and the control of oral anticoagulant therapy.[1] It measures the period required for clot formation when a subject's hemolyzed whole blood thromboplastin is added to his plasma. This test has the following advantages:

1) It is technically simple, rapid and inexpensive.
2) The patient's own hemolyzed red cells are used as the thromboplastin component eliminating the requirement of a foreign source.
3) It is sensitive to deficiences of all factors of both the extrinsic and intrinsic clotting systems (except platelets).

The end point for this procedure as in most clotting tests is the formation of fibrin fibers. Consequently, inter-observer differences occur due to slight variations in technical performance.[2] As automatic detection of fibrin formation would minimize this problem and increase its accuracy, we have adapted the ST-P time to an automatic prothrombin timer†. This paper describes the utilization of this instrument for the performance of the ST-P time and compares its reproducibility on duplicate plasma samples to that of one-stage prothrombin times in 100 patients on oral anticoagulant therapy.

Method

4.5 ml. of blood is drawn into a syringe containing 0.5 cc of sodium oxalate. 0.1 ml. of the oxalated whole blood is pipetted into a nonsiliconized test tube (8 cm in length, 1 cm in diameter) containing 2 mls of 0.02M $CaCl_2$ solution, and incubated in a 37°C water-bath for 10 minutes. This tube now contains recalcified whole blood hemolyzate which serves as the thromboplastin source for the test. The remaining portion of the blood is centrifuged for 5 minutes. When the recalcified hemolyzate has been incubated for 7 minutes two 0.2 ml samples of the patient's plasma are pipetted into sample vessels and placed in the slots of the dry rack heater. At the tenth incubation minute two 0.2 ml aliquots of hemolyzate are pipetted into the empty cavities of the sample vessels that contain the plasma samples. The vessels are then placed in the sample holder, the electrode bar inserted, and the machine started. The formation of fibrin is detected by the electrode bar which automatically stops the timer. The ST-P times are then converted to percentages by use of a dilution curve (Figure 1), obtained by the saline dilutions of pooled normal blood. For purpose of comparison Quick's one-stage prothrombin times were determined in duplicate on the same patient's plasma using Simplastin†† and were converted to percentages by the use of a prothrombin dilution curve for that specific thromboplastin.

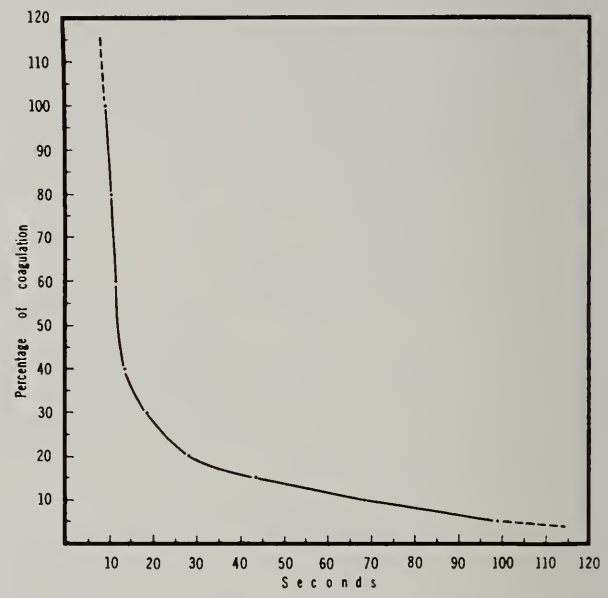


Fig. 1. The dilution curve of the ST-P time. The curve is prepared by the dilution of pooled oxalated whole blood obtained from normal subjects. Nine dilutions were made with whole blood concentrations ranging from 100 to 5%. The ST-P times were determined in triplicate for each dilution. A curve was drawn joining the plotted mean values. Points beyond the selected dilutions are depicted by dotted lines, and were determined by extrapolation.

Results

The results are tabulated in Table I.

Table I

	Self-Thromboplastin Prothrombin	*Quick's Time*
No variation between duplicates	25 cases	28 cases
Variation within 5%	47 cases	40 cases
Variation within 10%	17 cases	12 cases
Variation over 10%	11 cases	20 cases
Total	100	100

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Supported by Grant HTS-5475, National Institutes of Health

†Emdeco Prothrombin Timer manufactured by Electro-Mechanical Development Company, Houston, Texas
††Warner-Chilcott

Discussion

From an examination of Table I it would appear that the ST-P time is slightly more reproducible than the prothrombin time. This was found to be most apparent in the lower percentage ranges. Analysis of the groups with variation of over 10% revealed that the lowest result with the ST-P time was 36%, whereas with the prothrombin time there were 5 determinations with values below 30%. This is due to the fact that the prothrombin dilution curve is steeper for the lower percentages than the ST-P curve, and the differences in timing are magnified when converted to percentage. The advantages of a wider range in the lower percentages and a standard single dilution curve for the ST-P time has been discussed elsewhere[3] and is not within the scope of this paper. This preliminary study shows that the ST-P time can be easily performed on a prothrombin timer with the same precision as the one-stage prothrombin times.

Summary

This study describes the method for the performance of the self thromboplastin-prothrombin time (ST-P) on an automatic prothrombin timer. The reproducibility of the ST-P times and the Quick one-stage prothrombin times performed by this instrument on duplicate plasma samples is compared. The results demonstrate that the precision of the ST-P times on the automatic prothrombin timer is similar to that of the prothrombin times.

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Vesico-Ureteral Reflux in Children

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The subject of vesico-ureteral reflux and recurrent urinary infection is one of the most perplexing problems in pediatric urology. The normal physiology of the vesicoureteral junction is poorly understood. No one of the many theories offered by many authorities is universally accepted. Symptomatology as well as response to treatment in infants and young children is at best difficult to evaluate. By reason of their age, one has always to worry about the future of their renal function. By the same token, one wonders why this entity is not commonly seen in the adult population or, conversely, how many adult hypertensives and patients with chronic renal failure started life with unrecognized and therefore untreated vesico-ureteral reflux, especially before the days of improved radiodiagnosis.[1] Furthermore, it should be mentioned that though vesico-ureteral reflux is often coexistent with urinary infection, it is not always clear whether there exists a cause and effect relationship, and if so, which is cause and which effect.[2,3]

The object of this presentation is (1) to outline the case histories of a few representative patients who have come under our care in recent months; (2) to discuss the anatomy and pathophysiology of reflux; and (3) to give a brief summary of the various recommended methods of treatment.

Report Of Cases

1. M.M. (MMC #28672; Age 13 months). The patient first came under observation in April, 1964 in another city for a transient episode of fever with hematuria. Following a normal intravenous pyelogram, he was referred to the Maine Medical Center for further evaluation. Cine-cystourethrogram showed considerable reflux at rest up the right ureter. He was treated with Nitrofurantoin for three months. Reevaluation in November, 1964 showed a mild right ureteral atony by intravenous pyelogram with no evidence of reflux on cinecystourethrogram.

2. W.R. (MMC #31287, Age 3 years). The patient was first seen in February, 1964 for recurrent bouts of urinary infection, incontinence in spite of previous toilet training, and a past history of failure to thrive as an infant. Investigations showed bladder neck obstruction, a huge hydronephrotic nonfunctioning right kidney demonstrating vesico-ureteral reflux on cinecystourethrogram. He had a right nephro-ureterectomy in March, 1964. Follow-up studies in July after another bout of urinary infection revealed persistence of the bladder neck obstruction with reflux on voiding (not at rest) up the remaining left ureter. (Fig. 1) He had a Y-V plasty of the bladder neck in July. Reevaluation in November showed (Fig. 2) no bladder neck obstruction and no reflux. Clinically and bacteriologically he has remained free of infection. He was treated with Sulfisoxazole continuously until December, 1964.

3. J.M. (MMC #31562, Age 5 years). A mentally retarded boy from New Brunswick, Canada, was referred to the Maine Medical Center in September, 1963 having just recovered from a comatose uremic state with clinical evidence of chronic urinary retention. X-ray studies revealed no dye excretion from the left kidney, severe hydronephrosis on the right and an enlarged bladder. Cine-cystourethrogram revealed prompt low pressure reflux up the left ureter but not the right. (Fig. 3) There was also a suggestion of bladder neck obstruction.

On September 18, 1963, he had a preliminary decompressing suprapubic cystostomy. This produced sufficient clinical improvement so that on October 17, 1963, a Y-V plasty of his bladder neck was done with reimplantation of the left ureter.

As early as October 28, 1963, repeat intravenous pyelogram showed faint function on the left kidney after one

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Fig. 1, Case #2. Voiding cine-cystourethrogram. Note reflux up the ureter in the top right hand corner, and the sharp angle of the vesico-urethral junction.

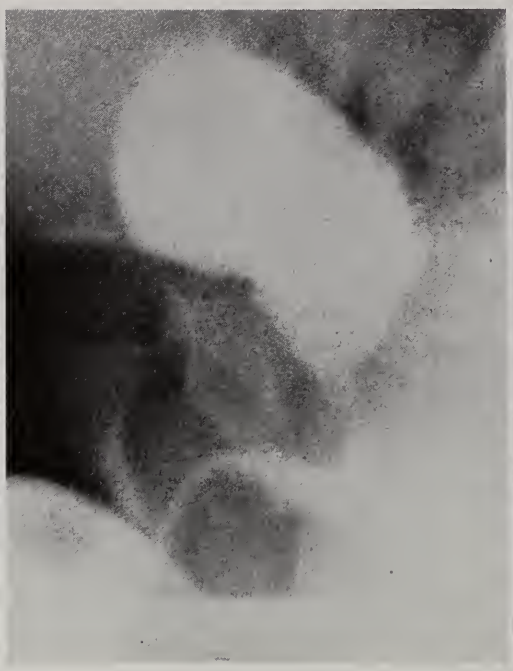


Fig. 2, Case #2. Voiding cine-cystourethrogram after Y-V plasty. Note the side funneling of the bladder neck and abscence of reflux.



Fig. 3, Case #3. Voiding cine-cystoureth gram. Note left renal pelvis filled by refl almost as big as the bladder.

hour. In April, 1964 dye excretion could be noticed on the left side at eight minutes. (Fig. 4) The right hydronephrosis appeared less than previously but cine-cystourethrogram now revealed reflux on voiding, but not at rest on the right side.

In November, 1964, although he had remained clinically improved, and repeated studies showed continued intravenous pyelographic improvement on the left side, cine study now revealed considerable reflux at rest on the right side and a degree of reflux on voiding on the left. Till the present he remains asymptomatic and is being followed on suppressive sulfisoxazole therapy.

4. R.D. (MMC #25818, Age 5 years). The patient was admitted to the Maine Medical Center in November, 1963 having been followed previously by a private physician. She had had five episodes of recurrent urinary tract infection, all responding temporarily to the usual drugs in adequate doses. Intravenous pyelogram at this stage showed good function, but cine-cystourethrogram showed gross low pressure bilateral vesico-ureteral reflux more pronounced on the left with good emptying of the bladder.

On November 5th she had a bilateral Leadbetter type ureteral reimplantation with a Y-V plasty of the bladder neck.

Clinically she did well, without any urinary infection, but her blood urea nitrogen in April, 1964 had risen from 24 mgs% to 49 mgs%. She was readmitted for reevaluation. X-ray studies showed uretero-caliectasis bilaterally. The ureteral orifices were dilated from size 4 F to size 7 F with ureteral catheters.

In September, 1964 repeat examination showed progression of the uretero-caliectasis in spite of an asymptomatic clinical course. (Fig. 5) She had an internal ureteral meatotomy and is currently being followed, and still asymptomatic.

5. R.M. (MMC #29446, Age 2 years). The patient first came under observation in September, 1964 with a two months history of dysuria, incontinence and enuresis. Investigations showed, besides pyuria and bacteriuria, a left double collecting system with uretero-caliectasis in the lower calyceal system. Cine-cystourethrographic study revealed low pressure reflux up the dilated ureter. Cystoscopy showed a normal right ureteral orifice. Of the two left ureteral orifices, the lower, more medial one, was normal and seemed to correspond to the right orifice, and the upper, more lateral one, was itself smaller than normal, but was situated at the inferior margin of a diverticulum about 1 cm. in diameter. Retrograde pyelograms showed this to be the orifice associated with the dilated collecting system. (Fig. 6)

Having failed to show any symptomatic improvement during three months of drug therapy, she underwent reimplantation of the refluxing ureter with a Y-V plasty of the bladder neck in December, 1964.

6. D.M. (MMC #29153, Age 9 years). This patient was referred to us in October, 1964. He had been healthy all his life apart from a few febrile illnesses. Three months prior to admission he developed a high fever with delirium. He was found to have a urinary infection which was subsequently adequately treated; but the entire picture recurred one month later at which time he was referred to us.

Our investigations revealed a positive urine culture for Aerobacter. An intravenous pyelogram showed bilateral hydronephrosis with the left kidney ectopically situated in the false pelvis. Cine-cystourethrogram showed a huge megacystic bladder, with severe vesico-ureteral reflux bilaterally at rest. (Fig. 7) The bladder emptied promptly. Both ureters showed good peristalsis and emptied their refluxed content back into the bladder filling it to a diameter of about 12 cm.

On cystoscopic examination there was no evidence of bladder neck obstruction. Both ureteric orifices were, however, rigidly patulous measuring 10. mm (definitely large enough to admit the tip of a Kelly clamp).[9]

It was thought that he should have a total urinary diversion or bilateral ureteral reimplantation. After considerable discussion, he is currently being followed with intermittent chemotherapy and frequent triple voidings, and has remained asymptomatic.

Comments

Each of these cases is distinctive and illustrates a particular aspect of this problem. The individual case histories hardly call for any comments except concern-



g. 4, Case #3. Intravenous pyelogram at 8 inutes. Note faint excretion of dye in the eviously non-functioning left kidney, six nths after Y-V plasty of the bladder neck d reimplantation of the left ureter.



Fig. 5, Case #4. Retrograde pyelogram. Ureters caliectasis bilaterally. Note the extreme narrowing of the distal ureters.



Fig. 6, Case #5. Retrograde pyelogram. The upper outer ureteral orifice drains the lower caliectatic renal pelvis.

ing the distinct significance of low pressure reflux versus the not too severe high pressure reflux, which seems nearly always to be secondary to bladder neck disease and to revert promptly when such disease is corrected.

Discussion

For further analysis of the problem as a whole, particularly the close association between the bladder neck and the vesico-ureteral junction, both in physiological function and in disease, the following brief review of the developmental and histological anatomy is offered.

Embryology

The ureteric bud (metanephric duct) is an outgrowth of the mesonephric duct, just cephalad to its opening into the vesico-urethral canal. The distal part of the mesonephric duct, called the common excretory duct, soon becomes absorbed and, at the 9 mm stage, both ducts open separately in the canal. Further development results in the migration of the metanephric duct opening cephalad and laterally. This migration is said to involve resorption of the anterior wall of the duct leaving the posterior wall behind as definite bands of mesodermal tissue extending from the site of the original opening (the definitive verumontanum) to the site of the final opening, the ureteric orifice.[5]

Histological Anatomy

In reviewing the histological anatomy of the ureterovesical junction, one must first say that, there is a lot of



Fig. 7, Case #6. Voiding cine-cystourethrogram. The bladder is huge, the left renal pelvis is just visible above the bladder dome. The right ureter is hydronephrotic and tortuous.

controversy here even in the more recent literature. Most of the controversy relates to "Waldeyer's Sheath" and its relation to the ureter in its intramural course. Another point of contention, and this is rather important, relates to the termination of the ureteric musculature itself. Woodbourne,[6] an anatomist at the University of

Michigan, maintains there is no such entity as Waldeyer's Sheath. He cites Waldeyer's original paper in 1892 and translates the original German to mean a "space" — an injectable space ½ to ¾ mm wide separating the ureter from the bladder musculature throughout its entire intramural course. His contention illustrates a common fault in eponymous nomenclature, but besides that does not mean much since he himself then goes on to describe a muscular investment of the terminal portion of the ureter which corresponds to what other anatomists describe as Waldeyer's Sheath. The terminal portion of the ureter seems to be broadly divided into three parts, juxta vesical, the intramural, and the submucosal portion. The juxta vesical ureter — mostly composed of longitudinal muscle fibres, but with obvious circular and oblique components like the rest of the ureter. It is invested with a muscular sheath, the identity of which is disputed, but which, however, is not a sphincter.[6,7] The remaining two parts (0.9 cm intramural, 0.7 cm submucosal) comprise of only longitudinal muscle fibres, lie free within an investment of Waldeyer's muscular sheath separated from the same by the injectable space described by Woodbourne. The muscular sheath itself is anchored to the vesical musculature around the inferomedial aspect of the hiatus.[7,8]

As regards the termination of the ureter and Waldeyer's Sheath, Tanagho differs from most other writers.[7] He maintains that at the ureteric orifice the longitudinal fibres forming the roof of the ureter diverge, sweep around the orifice and join the fibres of the floor to form a flat triangular sheet — the superficial trigonal muscle — most of the fibres of which find attachment to the verumontanum. The fibres of Waldeyer's muscular sheath do likewise, but the roof fibres diverge and flatten out at the junction of the intramural with the submucosal portion of the ureter. The triangular flat sheet so formed — the middle trigonal layer — inserts mostly at the bladder neck, but it is heavily reinforced by fibres from the deep trigonal layer which is formed by the bladder musculature itself.

Physiological Function

Many theories have been advanced to explain the still nebulous physiological function of the uretero-vesical junction: why efflux normally occurs but no reflux? Dr. Marshall summarized the prerequisite mechanisms as follows:[9]

(a) An adequate ratio of the length to the diameter of the intravesical part of the ureter. This normally is 6 or 7: 1; the critical ratio being 2 or 3: 1. When the ratio is less than this, reflux occurs.

(b) A posterior muscular support for the intravesical ureter. This is implied by the oblique course of this portion of the ureter.

(c) Flexibility: This seems to be the functional significance of the injectable space of Waldeyer, if by flexibility we imply mobility. It seems, as Tanagho points out, that this mobility enables the juxta-vesical portion of the ureter to be pulled intramurally during trigonal contraction or tension. Being thicker than the normally intramural portion, and being endowed with circular and oblique fibres, it constitutes an effective block to reflux during the phase of activity under consideration. It flexibility implies collapsibility then Marshall's analogy is self explanatory: "A steel pipe in there of any caliber and length might be expected to permit reflux."

(d) The functional possibilities of the anatomical set-up described by Tanagho might here be added as another possible theory[7] — namely the sphincter-like action of the ureteral muscle and the muscles of Waldeyer's sheath as they diverge to change from tubular structures to the flat sheets of the trigonal floor — the opening and closing movements at the ureteral orifice are often observed at cystoscopy.

Etiology Of Reflux

In discussing the etiology of reflux since there are so many possible theories, it is better in any particular case to talk about predisposing or associated clinico-pathological conditions than to talk about causes. Leadbetter categorizies these conditions as follows:

Bladder neck obstruction 50%
Neurogenic bladder — megacystis 30% — 40%
Cord bladder with obvious cord pathology 3% — 4%
Congenital anomalies of the junction itself, such as an ectopic ureteric orifice,[10] or a para-ureteric diverticulum.[8]

To these may be added two more groups. First is a small group in which transient reflux is associated with a transient attack of acute cystitis.[11] The reflux disappears when the infection clears. Another group may be called iatrogenic reflux; in these reflux follows surgery of the bladder neck or surgery on the ureterovesical junction itself.[7]

Management Of Reflux

The situation is well summed up by Politano and Harper as follows:[12] "Opinions regarding the management of vesicoureteral reflux are widely variant and range from the school of 'do nothing and let them outgrow it' to the school of 'reimplant them all.' In between are those who advocate prolonged use of bacteriostatic drugs, catheter drainage, urethral dilatation, transurethral resection, Y-V plasty of the vesical neck." (internal urethral meatotomy may be added to the list.) They then go on to describe long term follow-up (8 months to 16 years) of 27 patients treated conservatively with urethral dilatation or catheter drainage and long term antimicrobial drugs. They report symptomatic improvement in 1/3, but report complete disappearance of reflux in only two patients. Fifteen patients they claim had to have ureteral reimplantation for failure to improve, or for deterioration.

In a panel discussion, Dr. Stewart[9] seemed to stand "far out and alone in the middle of the very unpopular

and lonely desert of conservatism," to use his own words. He recognized bladder neck obstruction in 95% of cases and advocated surgical repair of the bladder neck by dilatation or what he described as "Crush-cutting punch by the suprabubic transversical approach". For those without bladder neck obstruction he proceeded as follows: "The patients are coached to avoid over-distention of the bladder and forceful voiding. They void every 1½ to 2 hours. At night they limit fluid intake and void once or twice." He condemned triple or multiple voiding as irrational, being designed to empty the bladder completely rather than prevent reflux. He also condemned the use of antibiotics prior to full urological work-up and correction of bladder neck obstruction if such there be. Even then, he would use antibiotics intermittently on clinical and bacteriological indications only. He regarded cholinergic drugs as of no benefit. He condemned any surgical interference on the uretero-vesical junction.

Among those who would operate, indications for operation seem to vary, at least in fine details:[9] Dr. Marshall would operate on all cases showing low pressure reflux especially when emptying of the refluxed ureters is impaired. He advocates bladder neck plasty as well as ureteral reimplantation in all such cases. In high pressure reflux, i.e., reflux on voiding but not at rest, he does only a bladder neck plasty.

Hutch seems to believe in eventual improvement in at least a percentage of cases. He would operate therefore only when he had to, basing his decision on the evidence of obvious hydroureter on intravenous pyelography.

Leadbetter would reimplant the ureters whenever reflux was well established and pronounced. He would also add bladder neck revision. He stresses the importance of preoperative evaluation of renal function. In poor risk cases he attempts to prognosticate by assessing the return of ureteral tone following a few days of urethral catheter drainage. He seems to have good faith in uretero-ileal diversions in children and would advocate this procedure in a few cases when he doubts the efficacy of reimplantation on the basis of too far advanced ureteral disease. Beyond a certain point even an ileal diversion might be hazardous in these patients.

At the Maine Medical Center there is no fixed doctrine of management of these patients. The tendency to date has been to assess each patient individually, bearing in mind, the age, the length of history, the family situation, the chances of follow-up, and last but not least, the anatomy of the defect. We have tended to offer antibiotics and bladder training as a first line of treatment. This has been difficult in many cases, particularly among the service-type patients. We believe that a bladder neck plasty should always be done whenever a ureteral reimplantation is done. Whenever urethral calibration reveals bladder neck obstruction, an internal urethrotomy has recently been adopted in place of Y-V plasty. Whenever deterioration is in evidence, particularly in the presence of bilateral renal disease or poor bladder function of a permanent type, urinary diversion is considered a sounder insurance against further deterioration than other less radical procedures. The earlier this procedure is done the better the overall result.

It seems difficult to escape the conclusion that the pathophysiology of the uretero-vesical junction involves the bladder neck, since both sites have such a close embryologic and histologic connection; in fact cine-radiographic studies both preoperatively and postoperatively in adults with bladder neck disease, such as prostatic enlargement, might well throw some light on the function of this anatomical complex.

Summary

Six representative cases of vesico-ureteral reflux are presented. The embryology, histology, and pathophysiology of the vesico-ureteral junction are discussed. The mechanisms for the prevention of reflux in normal function are listed together with conditions associated with derangement of these mechanisms. A review of the various methods of management of vesico-ureteral reflux is presented. It is suggested that cine-cystourethrographic studies in older patients with prostatism, pre and postoperatively might be illuminating.

Acknowledgment

Our thanks are due to the Attending Staff of the Urology Service for their painstaking supervision of the care of these patients, to Mrs. Gloria LePage for her help in preparing the original script, and Mr. James MacKenzie for preparing the illustrations.

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Report of Delegate to AMA House of Delegates Annual Meeting

Paul H. Pfeiffer, M.D.

The 1965 meeting of the House of Delegates of the AMA in New York City, June 20-24, was most interesting in that it allowed for a free and open discussion of many subjects, most importantly pending medical legislation called "medicare". During the first day of the session the hotel where we met was picketed by oldsters imploring the AMA "to give them a break on medicare." In the discussion widely divergent views were expressed. A strong, eloquent faction felt that the time had come for the medical profession to take a strong stand and block further trends towards the nationalization of medicine by non-participating in the anticipated federal medicare program. A similar move had apparently succeeded in Australia and led to the reversal of a National Medical Program sponsored by the Labor Government.

A more moderate view was expressed by the incoming President of the AMA, Dr. James Z. Appel, who took the stand that doctors had no right to civil disobedience and should comply with the laws of the land to the best of their ability. He regretted the fact that present trends sponsored by the Congress of the U. S. and the labor leaders were "making sheep" of the American people by imposing schemes for medical care on them which were to be payed for by taxes and allowed for no choice or development of individual sense of responsibility. Eventually Dr. Appel's point of view prevailed – against a strong undercurrent of opposition and after some harsh words had been said by the other side.

By electing Dr. Charles L. Hudson of Cleveland as the President-Elect it appears that the leadership of the AMA will continue to take a moderate tone.

112th Annual Session of the MMA House of Delegates

Sixty-one delegates and alternates registered for the 112th annual session at The Samoset, Rockland, Maine on Sunday, June 13, 1965. Linus J. Stitham, M.D. of Dover-Foxcroft, Speaker of the House, presided at the first session, which was called to order at 9:55 a.m. by the President-Elect, John F. Dougherty, M.D. of Bath. The Vice-Speaker of the House, Charles R. Glassmire, M.D., presided at the afternoon session.

Stenographic Record — A summary of the proceedings of the House of Delegates has been sent to the county secretaries and to the delegates and alternates of each county society and the complete Stenographic Record is on file in the Association office in Brunswick where it is available to any member of the Association. Consequently, this report will include only a few of the highlights of the meeting.

Maine Medical Education Foundation — It was voted that the active members of the Maine Medical Association be assessed $25.00 this year for the Foundation. Dr. Pfeiffer, Chairman of the Committee on Recruitment, Aid and Placement, commented on the "tremendous support from the Woman's Auxiliary." Dr. Hanley said that last year contributions from members of the Association combined with funds received from outside sources enabled the M.M.E.F. to grant financial assistance totaling $17,500.00 to twenty-three medical students.

Presentation of A. H. Robins Award — The Community Service Award for 1965 sponsored by the A. H. Robins Co. was presented to Niles L. Perkins, M.D. of Portland by Thomas A. Martin, M.D., MMA President.



Dr. Perkins receiving A. H. Robins Award from Dr. Martin

Medical School for Maine — Charles A. Hannigan, M.D. of Lewiston, Chairman of the Medical School for Maine Committee, reported that a State Legislative Order has been put through to investigate the possibility of having a Feasibility Study for a Medical School in Maine. He said that it would help if we could go on record as being fully in favor of this study and support it in any way possible and suggested that the House of Delegates vote a token sum of money to help with this program. This matter was referred to a Reference Committee consisting of Drs. George W. Bostwick, Chairman, Maurice Van Lonkhuyzen and Edward L. Reeves.

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Maine Medical Association

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DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

New Tools For Diabetes Instruction

RUTH T. CLOUGH, MS in HYG†

Recently, the Department has purchased four teaching machines primarily for professional and patient use in the area of diabetes instruction and review. The instrument selected is the AutoTutor Mark II, designed by Educational Service Division, U.S. Industries, Inc. Although teaching machines were first developed some 30 years ago, it is only in recent years that they have received sufficient attention to warrant and speed up their rapid development.

Extensive field investigation into the efficacy and use of the teaching machine has pointed up the fact that this tutorial method of teaching has several distinct advantages, basically as it concerns student learning: a) the student is required to respond continuously; b) he learns immediately whether or not his response is correct and c) he can progress at his own rate of speed.

In his paper prepared for the 59th Annual Health Conference, N.Y.* on the subject of the AutoTutor and its potential, Dr. Glen McDonald, Chief, Diabetes and Arthritis Program, Public Health Service has stated:

"We foresee a greater need for health education, especially for patients with chronic diseases. We will have to improve teaching methods to help these patients cope with a new situation in their lives – an ever-present disease condition. We must think in terms of life-long education; initially, to instill new facts, later to reinforce or update information. Physicians, also, will need new techniques to keep up with burgeoning medical research."

The machine under discussion uses the "branching" system of programmed learning. It is basically an automatic, semi-random access film projector. It provides training for the individual by selection and projection of separate 35 mm microfilmed images. Each reel of film stores 1500 – 5000 frames of instructional material. These reels can be readily changed thus allowing for a variety of different programs which may be presented through the same machine.

It is recognized that further studies will be needed to learn whether students are motivated to practice what they learn. However, from the field evaluations thus far made, investigators have been impressed with the opportunity given through this teaching medium for putting responsibility for self-care in the hands of the patient by requiring "him to press buttons and make choices." In addition, the patient is given opportunity to ask questions and because of the instant responses, his anxieties can be relieved; he himself intrinsically motivated.

Initially it is planned that the use of these machines will be directed to professional and patient groups in the field of diabetes instruction. Two films are immediately available for this purpose: *Diabetes Control* (a review course for physicians) and *Taking Care of Diabetes* (patient education and non-professional course.) The script for the former was written by Arthur Krosnick, M.D., a private practitioner specializing in diabetes, and Diabetes Coordinator for the New Jersey Department of Health. The course was programmed for automated instruction by a leading medical programmer, Miriam Hoglund Sierra-Franco. Physicians queried in respect to the machine by the U.S. Department of Health, Education and Welfare as well as The Medical Foundation of Boston which undertook a field evaluation under contract to help analyze the applicability and acceptability of the material on the professional course, have demonstrated considerable preference for this type of automated instruction over other methods such as lectures, workshops, medical books and journals, slides and tapes.

The script for the patient education film *Taking Care of Diabetes* was written under the sponsorship of the U.S. Public Health Service in cooperation with the American Diabetes and American Dietetic Associations, utilizing Dr. Krosnick and Norman Crowder as consultants. Materials covered in the film, in addition to introduction and general information, is as follows: occurrence, definition, symptoms, diagnosis and treatment; diet, food exchange lists, special dietetic considerations; medication (oral and insulin); use of hypodermic syringe, exercise, hygiene, foot care, urine tests, complications and conclusions. At the end of each section there

†Health Education Consultant
*June, 1963

is a quiz to test learning. The student cannot go on until he has answered each question and test correctly. The time required depends upon the student's background, reading and vocabulary skills, etc. However, the average time appears to take a person, moving at a reasonable rate of progress, between 2½ to 3 hours.

Other films available for use believed to be of interest to the physician and presently pending purchase are: "Body Fluid Metabolism," written by Arnold V. Wolfe, Ph.D., Professor of Physiology and Department Head, University of Illinois Medical College in conjunction with Norman Crowder; "Introduction to Asepsis," written by Marie M. Seedor, R.N., Ed.D., Instructor in the Department of Nursing Education, Teachers College, Columbia University. The course has been validated through the instruction of several groups of community college nursing students. Savings in class time over traditional teaching methods ranged from 9 – 65% with a medium time savings of 40%. When compared to students completing comparable classroom courses, the students using the AutoTutor teaching machine were found to have significantly higher achievement scores.

As time goes on, it is expected that the programming of desired subjects will increase rapidly. Suggestions from physicians in this respect are that cardiovascular disease, endocrinology, renal disease and liver systems might offer areas suitable for programmed instruction.

As to the current use of these machines, it is planned that they will be scheduled for placement, in accordance with and in order of requests, in suitable areas of hospital centers accessible to physician and patient respectively, thus to provide opportunity for physician review as well as for patient education, upon physician prescription for that purpose.

Another tool which has been found useful in patient education on the self-management of diabetes is the filmstrip-record series "Just One In A Crowd." This series of six, one-hour lessons was developed under the sponsorship of the U. S. Public Health Service and is designed to cover a six-week course for the diabetic and his family, under professional supervision. The course was recently given at Augusta General Hospital with marked success; is planned for further use incorporating the teaching machine at such points as may be feasible for introducing it to the physician, the patient, and the out-patient department of the local hospital where the course may be located. The filmstrip series covers the subject of symptoms, discovery and control of diabetes; is in color, and along with the desirable basic information indicated above, imparts the message that diabetics are "responsible people" – responsible for their own control, and, under the watchful eye of their physician, can learn to live and work to their fullest possible capacity.

The series is also available in 2 x 2 color slide sets with written scripts.

The above materials, filmstrip-record, slide-script series, are available to medical societies and hospital staffs for consideration in patient education programs. Scheduling and other arrangements for these, as well as requests for dietary consultation, can be provided by the Department for those who desire these services upon direct request to the Office of Health Education.

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3. McDonald, G. W. and Kaufman, Mildred – "Teaching Machines for Patients with Diabetes" – Journal of American Diet. Association – 42: 209-213, March 1963.

SPECIAL COMMITTEES 1965-1966 — *Continued from page* 205

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AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Smoking and Microscopy

Using techniques which enable them to see structures as small as about 20 billionths of an inch, scientists at Henry Ford Hospital have compared tissues removed from the lungs of bronchitic smokers and normal non-smokers.

The studies show that cells lining the breathing tubes of heavy cigarette smokers with chronic bronchitis were different in many ways from those of non-smokers; although the differences were only a matter of degree. Smokers' cells had changed, but they neither lost nor gained essential cell structures.

This work was described by the American Cancer Society in a report on Society-supported research by Drs. Geoffrey L. Brinkman of the Hospital's Pulmonary Division and John H. L. Watson of the Physics Department of the Edsel B. Ford Institute for Medical Research.

Samples of the bronchial lining were removed either by bronchoscope or during exploratory operations on the chest. They were immediately fixed, presumably in their natural form, and prepared for viewing in the electron microscope. The hardened samples were cut in slices about two millionths of an inch thick.

Under the electron microscope extremely fine structures within cell membranes and other cell constituents could be viewed clearly. Even large molecules could be distinguished over the vast landscape of the cell geography.

Of particular interest were 1) ciliated cells whose whiplike appendages serve as brooms in sweeping particles out of the breathing tubes, and 2) goblet cells which produce mucus that engulfs particles and allows them to be coughed up.

The tiny whips, called cilia, were of the same construction in the human bronchus as they are in protozoa and in other species. Each cilium in the broom was composed of nine double outer fibers and two single central fibers; a thin filament seemed to run through both central fibers. The cilia were anchored in basal bodies of the cytoplasm; bundles of fine fibers extended from the basal bodies to mitochondria, the cell powerhouses, and these fibers may serve as pipelines which transport the energy required to keep the brooms sweeping.

There were about 12,000 million cilia per square inch of bronchial lining.

Goblet cells were viewed in all stages of development. As they matured, they filled with mucus granules which pushed other cell structures aside.

These changes were noted in smokers' cells:

1) Projections of the cell protoplasm, which increase the cell's surface and possibly its ability to consume from the outside and secrete from the inside, were markedly reduced;

2) mitochondria were altered or underdeveloped, indicating subnormal power production;

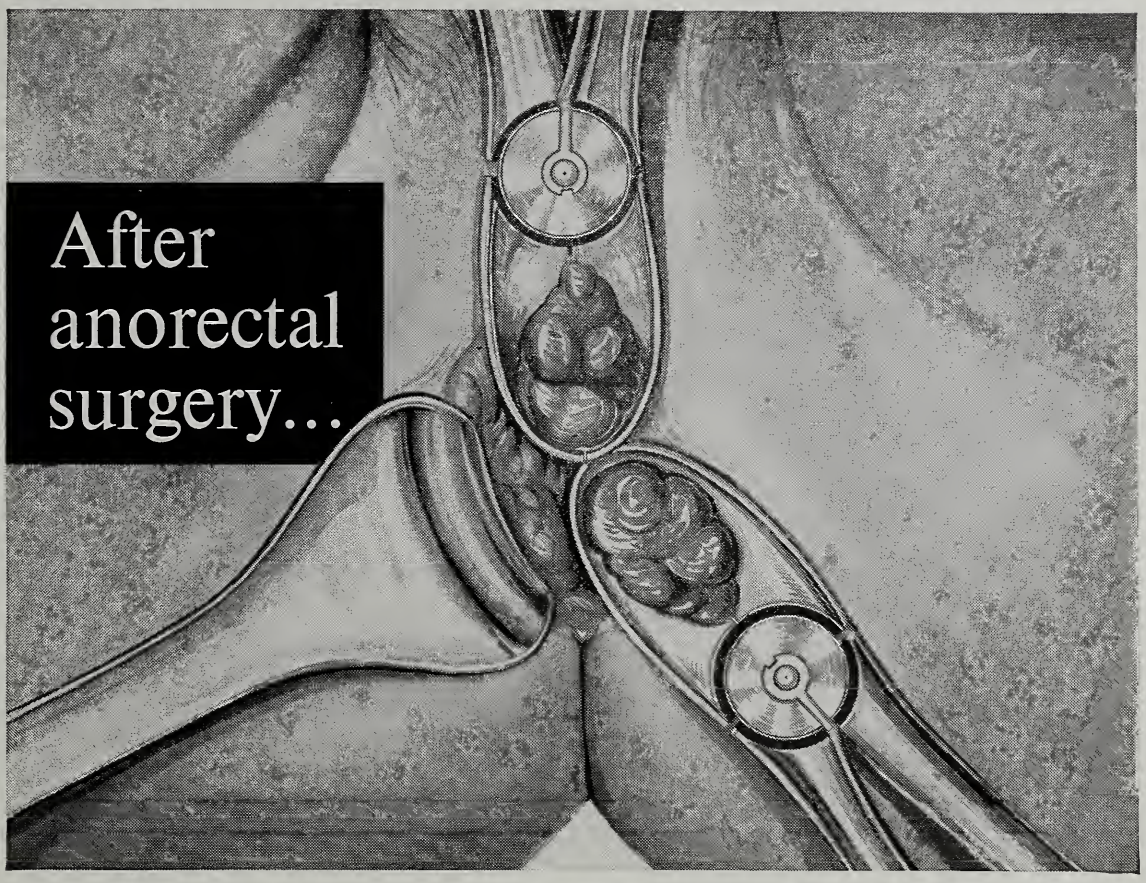
3) more frequent occurrence of what appeared to be secretion droplets;

4) more space between cells — possibly due to tissue water logging; and

5) more mature goblet cells, and increased frequency and size of fatty granules.

Smokers' cells appeared to have the normal number of cilia, and the internal structures of these whips seemed normal.

From American Cancer Society News Service Release



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THE PROBLEM of physical irritation of raw, postsurgical anorectal areas is frequently urgent. A well-verified satisfactory solution to it may be found in the bland, easily compressible "smoothage" of Metamucil.

The natural vegetable bulk of Metamucil softens intestinal contents and provides a soothing demulcent effect that protects denuded mucosal surfaces.

Metamucil prevents strain and traumatizing evacuations from constipation or cathartics and encourages the restoration of normal colonic function.

Average Adult Dosage: One rounded teaspoonful of Metamucil (or one packet of Instant Mix Metamucil) in a glass of cool liquid one to three times daily.
Metamucil is available as Metamucil powder in containers of 4, 8 and 16 ounces and as flavored Instant Mix Metamucil in cartons containing 16 and 30 single-dose packets.

SEARLE *Research in the Service of Medicine*

Maine Heart Association Notes

116 State Street, Augusta, Maine

The Incidence Of Arrhythmias In Acute Myocardial Infarction Studied With A Constant Monitoring System

"Death from acute myocardial infarction remains a challenging problem. Specific therapy is lacking, and treatment is largely directed to complications. One of the most disturbing of these is the occurrence of cardiac arrhythmias which may occur suddenly, without warning, and may remain undetected until harmful.

Our data confirm and extend the observations of others. . . . The longer period of observation permits us to conclude that significant arrhythmias may occur relatively late during the hospital course.

Although this series of 16 patients is too small to permit far-reaching conclusions, it strongly indicates that continuous cardiac monitoring . . . for a period of at least two weeks will reveal a much higher incidence of cardiac arrhythmias than previously reported in the literature. It also shows that arrhythmias often occur unassociated with clinical symptoms, and may go undetected by conventional clinical examination or serial electrocardiograms. Such arrhythmias pose a threat to life. Their early detection and treatment offer hope of reduced mortality. . . ."

KURLAND, G. S. and PRESSMAN, D., Circulation, Volume 31, Page 834-841, 1965

American Heart Association's Annual Meeting

The Annual Meeting and 38th Scientific Sessions of the American Heart Association will be held October 15-19, 1965 at the Americana Hotel, Miami Beach, Florida.

The Scientific Sessions are scheduled for October 15-17 and the Annual Meeting October 17-19. Advance registration forms can be obtained by writing the Maine Heart Association or the American Heart Association, 44 East 23rd Street, New York, N. Y., 10010.

112TH ANNUAL SESSION OF THE MMA HOUSE OF DELEGATES — *Continued from page 204*

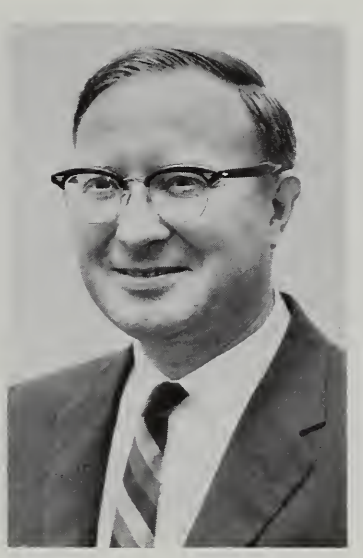
Their report at the second session of the House of Delegates which follows was approved.

1. That the House of Delegates empowers the Maine Medical Education Foundation to establish a separate fund to raise money for a Feasibility Study by a body acceptable to the Association of American Medical Colleges and the Committee on Medical Education of the American Medical Association, subject to provisions of the charter of the Foundation.
2. We further move that this body authorize the contribution of $2,500.00 to the Maine Medical Education Foundation towards this purpose.

Nominating Committee — The report of the Nominating Committee consisting of nominees for Standing Committees for 1965-1966 as presented by Charles R. Geer, M.D. of Portland, Nominating Committee Chairman, was approved. This report was published in the July issue of The Journal of the Maine Medical Association, page 168.

Budget for 1966 — The proposed budget for fiscal year 1966 as presented at the Interim Meeting of the House of Delegates by Paul S. Hill, Jr., M.D. of Saco, Chairman of the Budget Committee, was approved.

Re-Registration of Physicians — George E. Sullivan, M.D., Secretary of the State of Maine Board of Registration of Medicine, stated that in the last biennium, a re-registration law was passed. Beginning in July, 1966, at every biennium it will cost $5.00 for in-state physicians to be registered and $10.00 for out-of-state physicians.



DR. DENISON

Election of Councilors — Edward K. Morse, M.D. of Rockland was elected Councilor for the Third District (Knox, Lincoln-Sagadahoc Counties) and John D. Denison, M.D. of Gardiner, Councilor for the Fourth District

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ANDREW FERGUS, M.D.
Diplomate, American Board of Psychiatry
Chief of Staff

PHILIP BLINDER, M.D.
Psychiatrist
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(Kennebec, Somerset and Waldo Counties). Both of these appointments are for a three-year term. (Dr. Morse's photograph appeared in the August issue of the Journal.)

General Assembly

The General Assembly of the Maine Medical Association was called to order by the President, Thomas A. Martin, M.D. at 4:00 p.m. on Monday, June 14, 1965.

At this session, George E. Sullivan, M.D. of Fairfield, was elected President-Elect of the Association. Also, at this session Dr. Martin delivered his President's Address.

ESTHER M. KENNARD
Secretary-Treasurer, MMA

Announcement

Central Maine General Hospital

Hiebert Hall — 3:00-6:00 P.M.

Kidney Lecture Schedule — 1965

Oct. 27 Acid-Base Abnormalities in Hypercapnia and Investigation of the Patient with Kidney Stones
Jordan J. Cohen, M.D.

Nov. 3 Diagnosis of Kidney Disease and The Treatment of Acute Renal Failure
Jerome P. Kassirer, M.D.

Nov. 10 The Nephrotic Syndrome and Urine Cultures and Treatment of Urinary Tract Infection
Peter M. Berkman, M.D.

Nov. 17 Diuretic Therapy and The Treatment of Chronic Renal Failure
Norman G. Levinsky, M.D.

Nov. 24 Investigation of the Hypertensive Patient and Hyponatremia
Joseph A. Chazan, M.D.

Dec. 1 Treatment of Severe Hypertension. Treatment of Metabolic Alkalosis
Jerome P. Kassirer, M.D.

Fee: $30.00

Indications: Many types of edema involving retention of salt and water.
Contraindications: Hypersensitivity and most cases of severe renal or hepatic disease.
Precautions: Reduce dosage of concomitant antihypertensive agents by at least one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take special care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended.
Side Effects: Agranulocytosis, constipation, dizziness, dysuria, headache, hyperglycemia, hyperuricemia, impotence, leukopenia, muscle cramps, nausea, postural hypotension, purpura, thrombocytopenia, transient myopia, urticaria, vomiting and weakness.
Average Dosage: One tablet (100 mg.) daily with breakfast.
Availability: Tablets of 100 mg. in bottles of 100 and 1000.
For full details, see the complete prescribing information.

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The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, October, 1965 | No. 10

Trends In Medical Education*

J. M. Hayman, Jr., M.D.**

This is a time of great turmoil and unrest in medical education. There are three main reasons for this. First, the increasing knowledge of medicine by the lay public, and the consequent increased demands for more service as well as service of the highest quality. Second, the tremendous expansion of scientific knowledge in the last few years. Third, the beginning recognition of the importance of social sciences in the practice of medicine.

Just as fashions in dress have changed with the passage of time, so have patterns of medical care. Since World War II, medical science, medical education and medical care, each so directly related to the other, have been in a state of active ferment. Looking back over the past century one sees that medical care patterns have undergone profound changes, and that to a major degree these changes have already reflected the growth of medical knowledge and continued improvement in the education of physicians. From the days when medicine was almost entirely practiced by individual physicians dealing with an individual patient in his home, we have gradually moved to an era where in the cities, at least, the hospital assumed and has continued to carry an increasingly dominant role in the care of the sick. With this transition the character of the hospital itself has changed steadily and so has the pattern of disease that account for the majority of hospital admissions. The city physicians' dependence on the hospitals, on laboratory and on other physicians with specialized skill has grown apace. At the same time, rapid advances in many forms of therapy have been responsible for new emphasis on the ambulatory care of patients with illnesses that formerly caused them to be hospitalized. There have been, and are increasingly, changes in the method of medical practice, the development of group clinics and the development of prepayment and third party payment plans.

After World War II, two things happened. First, the Federal Government became deeply involved in the support of health related research in the Universities, and secondly, there was a jump in the birth rate from something like 19 to about 24 per thousand. These children born 20 odd years ago are now ready to enter medical school. Since the taxpayer was putting more and more money into medical schools by way of state and federal taxes, he expected to find the cause and cure of crippling diseases and to get more doctors to serve him.

Medical Schools are thus under pressure to do research, especially in the physical sciences, as well as to train doctors and also are under pressure to train more doctors and to teach more, especially in the area of genetics, behavioral sciences, bio-medical engineering, etc. Other areas whose proponents are demanding curricula time are psychosomatic medicine, comprehensive medicine, community medicine, general practice, clinical pharmacology, mental retardation, rehabilitation and molecular biology. If the efforts to introduce these new disciplines result in better teaching and research in classical departments, the goal of better education and patient care will be obtained. In the face of pressure to produce more and more doctors, it becomes more and more difficult to adequately train the present number. Are we approaching the situation faced here in the past, and in certain other countries of the world where the demand for more doctors will make us compromise with the quality of our education programs?

Changes in programs have been made. There has

*Delivered at 112th Annual Session, Maine Medical Association, June 14, 1965.

**Dean. Tufts University School of Medicine.

been a reduction in the number of lectures to allow more elective time which may be developed so that all students may not have to take the same courses. Programs in comprehensive medicine and home care, in the hope that the future doctor may make optimal use of his non-physician colleagues have been introduced. The study of the learning processes of teaching methods in medical education, as lead by Dr. George Miller, is under intensive study.

Another factor which is going to effect medical education is the very proper feeling of the practicing physician that he should look to the medical school or the medical center for help in keeping abreast of the expanding fund of knowledge and the more recent methods of treatment. The physician who has failed to maintain association with a medical college is in a waxing session of discontent. Advances and knowledge appearing at an almost explosive rate emphasizes the necessity for a solid background in social and other basic sciences, and the frightening technical complexities of modern diagnosis and treatment combine to make the conscientious physician feel that his grasp of the field is weakening and that he is losing command of the knowledge needed to know what is best for his patient. The physician is personally wretched because he cannot give that which he has for reasons conscious or unconscious pledged to give his patients, the best that modern skills have to offer the ailing man. This is most true of the man who attempts the most herculean of all medical tasks – general practice. But it is also true of the specialist who, for reasons personal or geographical, loses regular contact with the teaching institution. The solo practitioner in a particular field finds himself challenged by his confreres in the great medical centers of the land, by experts in the allied health professions, and even by those patients who read magazines and health articles and the titillating medical columns in the daily press.

The American Academy of General Practice, so far as I know, is the only professional organization that has fully realized this necessity and that requires continuous study by its members in order to maintain membership.

The great change in medical education, however, should be in the training of personal physicians because the general practitioner of the past, in the sense of the man who did some surgery, some obstetrics, some psychiatry, and some emergency work, is no longer filling a need in most communities. But his place as personal medical attendant must be filled. There is almost universal accessibility in this country to surgeons and other specialists, and there is no reason for general practitioners to attempt to do work for which they are not really trained. However, there is still the greatest demand for personal medical attendance and, therefore, immediate and careful studies should be given to the problem of the preparation of personal physicians.

The importance and status of the family physician must be increased. His income should be as high as any in the medical field. His qualifications should be carefully studied and his training carefully planned. It has been proposed that certain schools specialize in the production of family physicians, for these are needed in solo, group, and institutional practice in very large numbers, and soon, while other schools should train specialists and investigators.

I do not believe, however, that there should be two kinds of schools. The personal physician or the man in solo practice needs, to my mind, an even better training than the specialist. It is he who sees the patient first and must make his decisions on incomplete data, using his head which is, as has been pointed out, the only computer that can be mass produced by unskilled labor. The specialist, on the other hand, can demand all kinds of laboratory procedures and simply sit in his office until these are all in and the diagnosis handed to him by the computer.

Most schools are attempting to develop curricula that discourage encyclopediac memorization of facts, but stress how to learn and how to use learning, rather than what to learn and how to pass examinations. The new look in medical education calls for the development of teaching and learning methods that are calculated to inculcate in the student the ability to select, organize and evaluate information so as to stimulate as well as to satisfy curiosity, and to foster the kind of critical perception and sensitivity that the judgments of medicines increasing effectiveness will require. In short, the medical school attempts to educate an undifferentiated physician whose differentiation and training for the type of practice of his own choosing will take place during his years of internship and residency.

In the United States the number of those over 65 years of age have quadrupled. There is less acute and infectious disease and more chronic disease, less death and more disability. In chronic disease there is less present possibility of cure and greater need for prevention and early diagnosis. While there is increasing reliance on technical hospital base procedures there is an increasing need for comprehensive therapies that are more than narrowly medical and involve many disciplines, many facilities and many settings, including the patient's family and community.

Unfortunately, the way that our medical teaching is organized at the present time, there is too much arbitrary separation of the acutely ill, the chronically ill and the mentally ill patients without regard for their real needs for care and at a staggering cost in duplication of staff and facilities. It is almost impossible for the student to follow his patient out of the hospital and into home and community. Much of the young physician's subsequent experience in practice will be in ambulatory medicine, but the facilities in the academic environments of the out-patient department and the emergency ward in which he works during his training are often totally inadequate, isolated both from inner hospital experience and from the community.

In the confines of the ward and laboratory the student has little opportunity to explore the influences of social behavior and environmental factors in the ideology of the therapy of disease.

It has been suggested that to meet this dilemma the medical school should undertake research and experimentation in medical care and service. It is suggested that the medical school should get involved in various forms of medical care such as group practice, or in assuming the total responsibility for a whole group through affiliation with labor groups, prepayment schemes and the like, or by simply accepting responsibility for all medical services for an entire community or for some defined population or area within it. It is argued that thus they will simultaneously be able to explore and demonstrate to students and the profession the patterns of practice most consistent with current medical knowledge and to promote neglected subjects and methods of research.

However, these proposals are not universally accepted by any means. In the first place they might well bring the medical school into even greater conflict with the profession, community hospitals and with organized medicine. Secondly, and a very real point, there is a question of whether such assumption of service responsibilities should be classed as educational. Medical schools, Dr. Dixon observed, are not now equipped to teach the student how to administer medical care or how to deal with its economic, political and social aspects. Whether this is an educational objective is debatable.

We all give at least lip service to the essential role of the family physician and to the idea that he must be a key figure in the medical practice of the future. Yet, unfortunately, we do not really know what the family physician is now, nor haw he should be trained for the future, nor how the public regards him. Friedson has recently provided evidence that the idea of a family physician is pretty much an article of faith, operating powerfully in the evaluation of physicians even by those patients who have never had a family doctor. Although better than 75% of United State residents in sample after sample have said "yes" when asked if they have a family doctor, a high proportion of these same respondents also report that they regularly consult pediatricians, obstetricians and other specialists. There is at present considerable cloudiness as to what the function of the family physician should be. We are told that he will "see the patient first and be a diagnostician, evaluator, an analyist of data, a counsellor of the patient relying heavily on specialists." Or again, his function will be the general supervision of health and medical care preventive medicine, the treatment of minor illnesses and the practice of minor surgery and normal obstetrics the screening of patients for early manifestation of severe and chronic illness. It is said that he will assume comprehensive and continuing responsibility for the patient and his family regardless of age, although his training will be sufficient only to enable him to understand "the general diagnostic and therapeutic principles." He will "manage chronic illness and rehabilitation"; he will be a diagnostician and a health counsellor who will promote health.

It is suggested that his training should be divested of scientific details and too much exposure to the basic medical disciplines. Yet he is supposed to have enhanced clinical acumen in the early diagnosis of chronic illness – certainly the most difficult problem with which a physician can be faced. It seems to me that all of this represents confused thinking.

Dr. Cecil Sheps has emphasized that the increase of chronic illness has had a profound influence on the nature of the physician's task. While diagnosis he admits, is still important, interesting and sometimes difficult, the crucial problem is often that of treatment and the problem of treatment is increasingly one of how to work with the patient, his family and his community so that he can function as effectively as possible within the framework of his disability. Moreover, treatment frequently requires modifying the patient's habits and attitudes and bringing the resources of the community into focus in order to deal effectively with his particular problem. The increasing prevalence and the prominence of chronic disease, therefore, highlights the importance of comprehensive medical care. To understand his patient, the physician must know the natural history of chronic illness. Optimum care is often conditional upon the understanding and support of the family and community. The resources that are needed frequently involve the skills of other professions and community facilities, agencies and programs.

Various attempts are also being made in a number of schools to teach what has come to be called comprehensive medical care. At Tufts, fourth year students spend two months on the home medical service and in the out-patient clinics where they visit both acute and chronic patients in the home, and see them when necessary in their offices in the out-patient department. I may be prejudiced, but to my mind this is the best attempt to provide experience in family practice under supervision that is available.

Certainly the extent of specialization has increased and this trend can be expected to continue. At present some 85% of graduates specialize and the decision is usually made early. Since with this trend physicians in specialty practice will commonly be the physicians of initial contact, they must be prepared to assume a general responsibility for defining the health needs of the patient. Thus, there seems to be the paradox that specialists will carry increasing responsibility for general health care, while the generalist is forced to limit the variety of his clinical responsibility.

The rise in specialization has not resulted from the actions of physicians alone. The public has equal responsibility. As health sophistication and the ability to pay have increased, people want the best, which they equate with specialists. While a man wants a family

doctor near at hand and on call, when he is really sick, or wants a complete "check-up," he heads for the specialist or clinic.

I have tried to outline for you some of the problems and pressures that are being discussed at the present time. The essential elements, I believe, are that there are not enough physicians, that these are not equitably distributed, that there has been an explosive increase in medical knowledge and technology, and that the public is demanding more and "better" care and that the medical schools are being asked to correct all these defects.

Dr. Coggeshall has urged the AAMC to take a more active role in preprofessional, professional and postgraduate programs. He believes it should give particular attention to developing improvements in the team approach in both hospital care and medical practice. But at the same time, aid should be given in development of improved approaches to family practice.

My concern tonight, perhaps a narrow one, is to indicate to you the trends I forsee in the medical program itself.

First the selection of students. We must try to develop better methods of judging motivation – of identifying applicants with the intellectual and moral integrity we all want in the profession, and a real desire to be of service and to contribute to the health care of the people. We should not be influenced entirely by high aptitude tests and high college record. At the same time we must avoid accepting applicants, no matter how well motivated, who do not have the intellectual capacity to meet the rigors of the medical curriculum. For the increase in scientific knowledge demands a much more thorough understanding of biochemistry, genetics, microbiology and biophysics than was required when we went to school if presently available methods of diagnosis and treatment are to be made intelligently available to our patients.

We must give up the notion that on graduation the students knows all the medicine he will ever need to know. We must abandon stress on the memorization of facts for training in the methods of self-education. We must certainly pay more attention to the ecology of ill health, to the roles of environment, emotional factors and ethnic customs. We must emphasize that man is more than the "ingenious assemblage of portable plumbing" described by Chris Morley.

We must provide more training in the problems of geriatrics, in preventive medicine, not in the sense of preventing epidemics of contagious disease, but in preventing or slowing up the development of incapacity from chronic disease. We must certainly put much more emphasis on rehabilitation, an exciting and sadly neglected field.

The student must know something of the facilities available to provide medical care – of the role of the dentist, nurse, physical and occupational therapist and clinical psychologist – and how as leader of the health team he can mobilize disciplines for the care of his patient. Even before he leaves medical school he should know something of the techniques, advantages and disadvantages of different types of practice – institutional, group, solo, and the differences in urban and rural practice.

The importance, scope, and dignity of family practice must be re-oriented. The fake idea that the family doctor is second-class, and is just a sorter for specialists must be abandoned. He can take care of 95% or more of his patients, provided he uses the knowledge and skills which he should have. To refer a patient to a surgeon for an appendectomy is less evidence of lack of competency than for the surgeon to refer a patient to an otolaryngologist to have his ears washed out or for the internist to refer one to a surgeon for a proctoscopic examination.

Merely increasing the number of physicians graduated is not going to solve the problem of distribution. They will still tend to concentrate in the cities unless rural practice can be made more attractive. This to my mind is primarily a problem for the county medical society rather than for the medical school.

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Peritoneal Dialysis

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The standardization and wider use of peritoneal dialysis has followed the progressive understanding of renal insufficiency and uremia as reversible and potentially curable. This article will describe the present status of this technique as usually applied to patients with acute reversible or borderline chronic renal disease, as well as poisonings, and other non-renal problems. The following case history will illustrate the use of peritoneal dialysis (and hemodialysis) in acute tubular necrosis.

Case History

R. B. This 28-year-old man had been caught between a tractor and a rock trailer sustaining a severe crushing injury of the right upper thigh and abdomen. He had gross hematuria initially followed by oliguria (100 to 125 ml of urinary output per 24 hours). He was first seen at this hospital two days following the accident. He was alert but vomiting guaiac positive material. The abdomen was diffusely tender, tympanitic, and quiet on auscultation. The right thigh was swollen to twice its normal size, had a purplish discoloration and large areas of blistered and questionably viable skin. The pulse was weakly palpable below. The BUN was 73 mgm % and urine urea concentration 140 mgm %.

He was treated with Levine Tube drainage, 800 ml of intravenous 20% dextrose in water over each 24 hours with insulin to combat hyperkalemia and sodium bicarbonate for metabolic acidosis. The third day following admission he had become slightly confused. The BUN was 137 mgm % and serum potassium 7.1 meq/L with ECG showing signs of hyperkalemia. Because of extensive previous abdominal surgery (bowel resection, colostomy, and cystotomy ten years prior to this illness) and because of possible bowel injury from the accident, hemodialysis rather than peritoneal dialysis was undertaken. Six hours of dialysis corrected the confused mental state, lowered the BUN to less than 85 mgms % and returned the potassium values to normal. Cystoscopy revealed enlarged kidneys and no obstruction. A renal scan with Hg_{205} showed poor uptake bilaterally. The abdominal tenderness had resolved and the levine tube was removed. However, because of possible bowel injury the oral administration of exchange resins to control hyperkalemia was not initiated. The patient appeared well though still oliguric. On the seventh hospital day the BUN was reported as 257 mgm % and serum potassium 8.7 meq/L. He was taken to the operating room and a peritoneal catheter inserted under direct vision dividing several omental and bowel adhesions to place the catheter from the right abdominal wall into the pelvis.

The BUN fell to 160 mgms % after 36 hours of peritoneal dialysis. He started taking a 20 gram protein diet by mouth the day following insertion of the catheter. The peritoneal dialysis was discontinued after seven days (16 days after trauma) at which time the BUN was 138 mgm % and urinary output 600 ml/24 hours. Though the BUN rose again to 174 mgm % the patient was clinically well. The leg injuries improved and he was discharged on the thirty-third hospital day (with a BUN of 25 mgms %).

Comment: This case demonstrates the rapidly progressive uremia and hyperkalemia associated with trauma, and the ability to control it with peritoneal dialysis. Since this patient was treated, renal biopsy has become a standard and early part of the management of acute renal failure.

History

In 1876, Wegner, while studying postoperative shock, found the peritoneum to be a semipermeable membrane and performed peritoneal infusions on experimental animals and humans.[1]

Ganter, in 1923, performed the first successful peritoneal dialysis on anuric rabbits, but failed in the first clinical dialysis in the treatment of chronic nephritis.[2] Darrow and Yannet, in 1935, established an important landmark by demonstrating the fluid and electrolyte shifts between intracellular and extracellular compartments.[3] This knowledge was applied by Frank Seligman and Fine, who in 1946, performed the first successful continuous peritoneal dialysis.[4] They were, however, confronted repeatedly with the problems of over hydration, peritonitis, obstructed catheters and hyperchloremic acidosis. The dialyzing solutions were hypotonic and contained excessive amounts of chloride. Large sump drains were easily plugged with omentum and fibrin clots. Broad spectrum antibiotics were not available. With repeated clinical failures, peritoneal dialysis fell into disrepute. When, in 1947, Kloff demonstrated the clinical applicability of the artificial kidney, clinicians were led further away from the peritoneal approach.[5]

It was not until 1951 that Grollman, Turner and McLean performed the first truly successful peritoneal dialysis employing a closed intermittent dialysis with hypertonic solutions containing sodium lactate and prophylactic antibiotics.[6]

Maxwell, in 1959, devised the technique currently in use by refining the method of Grollman.[7] This method uses intermittent dialysis, limited to approximately 36 hours. Now broad spectrum antibiotics are used prophylactically; newly designed plastic catheters have overcome the problem of plugging by fibrin, and hypertonic dialyzing solutions, with more physiologic electrolyte compositions, have become commercially available.

Peritoneal dialysis and extracorporeal hemodialysis, now established procedures in the medical armanentarium, have come to complement each other. Choice of procedure is now based on proper patient selection and availability of facilities.

Mechanisms In Peritoneal Dialysis

The Peritoneum. The peritoneum has a surface area

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Table 1

Ion Content Of Body Fluids Compared To Dialyzing Solutions (meq./l)

Solute	*Plasma*	*Interstitial*	*Dialysate*
Na+	142	146	140.5
K+	5	4	(4) *
Ca++	5	2.5	3.5
Mg++	3		1.5
H_2CO_3	1.4	1.4	
pH	7.4	7.4	5.0
Cl	103	115	101
HCO_3	27	30	(44.5) *
Protein	16	1	
Org. Acid	5		
$HPO_4^=$	2	2	
$SO_4^=$	1	1	
Lactate			44.5
Dextrose			15 GM/L. (70 Gm/L.) *
mOsm/L	310	310	372 (1.5% Dextrose) 677 (7.0% Dextrose)

*optional additions or substitutions.

of 22,000 square centimeters in the adult. This approximates the 15,000 square centimeters available for glomerular filtration in the kidney. The peritoneum acts as an inert semipermeable membrane performing no active transport. It permits the passive movements of water, electrolytes, glucose, urea, amino acids, non-aminated organic acids and dialyzable poisons. Direction and rate of diffusion of these solutes across the peritoneum is determined by individual concentration gradients. Diffusion of water is determined by the osmotic gradient.

Dialyzing Solutions. Commercial dialyzing solutions* are compounded to be in chemical equilibrium but hypertonic to normal plasma and extracellular fluid (See Table 1.) They have an electrolyte concentration equal to normal plasma, thereby avoiding alterations in normal plasma electrolytes. The solutions commercially available are essentially extracellular fluid without protein and with enough dextrose to increase osmolarity to 372 milliosmols per liter rather than the 290-310 milliosmols of normal serum. A slight osmolar excess prevents absorption of fluid from the peritoneal cavity and the over-hydration previously encountered in uremic patients treated with isomolar solutions. Uremic serum is usually hypertonic due to retained metabolites. During dialysis the patient's extracellular electrolytes and metabolites whether high or low in concentration will shift toward a concentration of the dialyzing solutions. Hyperosmolar solutions draw water into the peritoneal cavity. Poisons, excessive metabolites, and electrolytes will diffuse into the peritoneal fluid.

There is usually no need for alterations in the electrolyte composition of the commercial solutions. This may actually be hazardous and contributes little to the overall clinical results. Potassium is deleted from these solutions that are used in hyperkalemic patients. However, in dialyzing hypokalemic or normokalemic patients, potassium chloride 4 mEq must be added to each liter of dialyzing solution. Calcium ion is maintained at 4 mEq per liter, slightly higher than the normal ionized fraction of serum calcium. Magnesium is maintained at the safe lower level of normal (1.5 mEq per liter).

Doolan and others have performed successful dialyses by instilling, premixed, the following commonly available solutions: 1 liter 5% D & W; 1 liter 5% D & S; 1 liter normal saline; sodium bicarbonate 7.5 grams in 100 cc. volume. The net result of such a mixture is a volume of 3,100 cc with the concentrations of sodium 128 mEq. per liter, chloride 100 mEq per liter, bicarbonate 28 mEq per liter, glucose 32.5 grams per liter, a pH of 8.0, and an osmolality of 400 milliosmols per liter.

A number of agents are usually added to the dialyzing solution. Heparin is necessary to prevent obstruction of catheters by fibrin and blood clots. Unless there is a specific contraindication, 50 units (5 milligrams) per liter may be added. Xylocaine® 0.5 cc of 1% solution may be added to relieve abdominal discomfort caused by dialysis. Albumin added to the dialyzing solution results in an increase of 80% (40-140%) in the binding and removal of protein-bound poisons, such as barbituates, salicylates and bilirubin.[8,9,10,11,12] Human albumin may be added to make a 5% solution. This is also commercially available.† Albumin is also recommended as an additive to prevent further protein loss by dialysis in severely hypoproteinemic patients. Dextrose may be added to increase the osmotic pressure of dialyzing solutions to prevent the retention of water. Each one and a half percent increase in dextrose concentration raises the solute concentration by 82 milliosmoles. Dextrose 52 to 200 cc of 50% solution may be added as needed to as many as four consequetive lavages. Alternatively, one may use prepared solutions containing 7% dextrose per liter. Plasma or albumin may be required intravenously to support circulating blood volume during the use of these hyperosmolar solutions. Antibiotics are added to treat or as prophylaxis against peritonitis (See complications).

Technique Of Peritoneal Dialysis

Important in the initial management of dialysis are accurate clinical and laboratory base lines for following the patient's progress. Thereafter, the following data should be charted at regular intervals. An hour to hour check on fluid balance is essential to a safe and efficient dialysis. With each lavage record input and output, changes in clinical picture, vital signs, drugs added to the dialyzing solution and note adjunctive therapy. Weight should be recorded at least daily or more frequently as a check on water balance. Serum electrolytes are obtained daily. Cultures of the dialysate are taken every third day. The electrocardiogram may serve as a guide to managing problems involving serum potassium and calcium.

*Impersol (Baxter Laboratories, North Chicago, Illinois)
†Albumisol-Merck Sharpe and Dohme, Westpoint, Pennsylvania

Peritoneal Catheterization

Catheterization is usually accomplished through a paracentesis or gallbladder trochar, however, a surgical incision offers the safest approach in the presence of bowel distention or if postoperative adhesions are suspected. Successful dialyses have been performed on infants using only a large lumbar puncture needle. If chronic dialysis is anticipated one may employ a permanent peritoneal access prostheses ("button").[13]

Before catheterization is started the colon should be emptied by enema and the bladder drained by catheter to avoid penetration of viscera. The stomach should be emptied initially by Levine tube to avoid aspiration which may be precipitated by intraperitoneal infusion (particularly in the comatose patient).

Before inserting the torchar into the peritoneal cavity, especially if there is underlying bowel distention, it may be safer to initially distend the peritoneal cavity with one or two liters of dialyzing solution using a short tapered #16 needle. The site of penetration is preferably along the linear alba, one third the distance down from the umbilicus to the pubic symphysis. The right and left lower quadrants lateral to the inferior epigastric vessels are the best alternate sites. The skin is incised with a #11 Bard-Parker blade that is advanced to the fibrous linea, identified by a grating sensation. If bleeding developing at the puncture is refractory to simple pressure, one may convert the site into an incision to allow direct hemostatic control. A #16 to 24 Ochsner gallbladder trochar is then forcefully pressed through the linea and peritoneaum (at a right angle to the skin). A #11 French plastic dialyzing catheter, rinsed in heparin is then passed. As an emergency substitute one may employ a new plastic gastric suction tube with 3/16th holes cut in its distal 8 inches. The catheter is advanced into the pelvic gutter. If difficulty is encountered in positioning the catheter tip, accurate placement may be obtained after the abdomen has been distended with dialyzing fluid. To avoid local edema and loss of fluid one must be certain that the catheter perforations are deep to the peritoneum. A nylon purse-string suture is then placed in the skin and subcutaneous tissues about the catheter to avoid leakage and infection. The site is sterilely dressed and cleansed daily with strict aseptic technique. Leakage of dialyzing fluid through postoperative wounds is not an uncommon problem, readily correctable by reinforcement with additional sutures. Thereafter, healing proceeds normally.

Peritoneal Lavage

The Infusion. Dialyzing fluid is preheated to 40°C. With a three foot gradient from bottle to patient, a minimum of 10 minutes is allowed for inflow. Infants easily tolerate 75 to 100 cc per kilogram of body weight. Children over five years of age tolerate 2,000 cc total quite well.[14,15,16,17] A useful formula for calculating the volume is 1200 cc per square meter of body surface area. Excessive volume may cause dyspnea. Anxious patients may require sedation. A mild abdominal discomfort due to chemical irritation may warrant the addition of Xylocaine to the dialyzing solution.

Table 2

Antibiotics In Peritoneal Dialysis
Dosage per liter dialyzing solution
Tetracycline 12.5 mg.
Penicillin 5,000 to 200,000 units
Streptomycin 25 mg.
Sulfadiazine 60 mg.
Kanamycin 50 mg.
Cephalosporin 50 mg.
Neomycin 2 mg.
Polymyxin 5 mg.

The Dialysis. The greatest exchange of free ions and metabolites across the peritoneum is complete in one hour. Maximum exchange is obtained in two hours. The average time for efficient dialysis of these ions ranges from 60 to 90 minutes. The most efficient removal of protein bound poisons is accomplished after 90 minutes of dialysis with solutions containing albumin.[9,10,11,12]

Drainage. If drainage time exceeds 15 minutes or the returning volume is diminishing, one should suspect obstruction of the catheter by fibrin, blood clots, or omentum or by kinking of the catheter. Careful manipulation, irrigation, or replacement of the catheter is indicated. Drainage fluid is clear to opalescent yellow. The bloody color of dialyzing fluid in uremic patients clears as the BUN decreases. The smear of drained fluid frequently demonstrates 10 to 400 leucocytes per HPF. The protein content averages 50 mg% (50 to 150 mg %). Simultaneous samples of dialyzing fluid and serum have identical concentrations of electrolytes.

Extended Peritoneal Dialysis. The average procedure ranges from 12 to 36 hours employing 30 to 50 liters of fluid. The procedure may be interrupted to allow ambulation. A single well-functioning catheter may be left in position over a week. Ileus is common during the first 48-72 hours of dialysis, but this usually remits spontaneously and is treated by restriction of oral intake and Waggenstein suction. Selected cases of chronic renal failure with oliguria may require dialyses two to six times weekly employing 10 to 70 liters of dialyzing fluid according to the given patient's metabolic needs as governed by his renal function, general condition, and diet.[18,19,20]

Complications In Peritoneal Dialysis

Peritonitis. This problem has been largely eliminated by strict adherence to aspectic technique, the use of closed sterile system of lavage and the prophylactic use of broad spectrum antibiotics. Cultures of the fluid are occasionally positive in the absence of clinically detectable peritonitis. Actually peritonitis is no longer a contraindication to dialysis. Dialysis need not be stopped in the presence of acute peritonitis. The specific organism should be treated with the proper antibiotic

based on sensitivity studies. Current authorities on the subject are divided between the group that prefer to employ a broad spectrum antibiotic prophylactically,[7,18,19,20,21] and the group that employ antibiotics only when peritonitis is definitely present.[15,16] At the Maine Medical Center, tetracyline has been the preferred antibiotic employed in dialyzing solutions (with and without simultaneous systemic administration of antibiotics). See Table 2 for list of other antibiotics recommended in the current literature.

Hypoproteinemic Edema. Hypoproteinemic edema becomes a problem during long-term or repeated dialysis, since up to 20 or 30 grams of albumin may be lost in 24 hours of dialyses. Correction is by intravenous administration of plasma or albumin, especially in patients with initially low serum albumin. Losses of albumin are lessened by adding albumin to the dialyzing solution.

Lacticacidemia and Metabolic Alkalosis. Lacticacedemia may develop during dialysis with advanced uremia or liver failure as a result of the decreased conversion of the lactate ion from dialyzing solutions (45 meq/1.) to bicarbonate ion. Metabolic acidosis is then improved by the intravenous administration of sodium bicarbonate or by using Doolan's solution containing bicarbonate ions (28 meq/1).[8] Metabolic alkalosis seldom occurs with modern solutions currently used and rarely is an indication for any therapy.

Extraperitoneal Dissection of Dialyzing Fluid. Mediastinal edema may slowly develop during dialysis and should be suspected if blood pressure slowly falls or dyspnea occurs. This may be corrected simply by elevating the patient's head or assuming a sitting position. *Hydrothorax* may develop suddenly and be heralded by acute onset of dyspnea in a patient who has sustained tears of the diaphragm through trauma. This may respond to surgical correction. *Retroperitoneal dissection* may develop in the same manner following resection of abdominal aortic aneurysm. If the above corrective measures fail, dialysis must be accomplished by hemodialysis.

Indications For Peritoneal Dialysis

Acute renal failure. Indications for dialysis may be defined as: a BUN above 150 milligrams percent associated with a serum potassium approaching 7 milliequivalents per liter; a serum bicarbonate below 12 milliequivalents per liter, pulmonary edema, or deterioration of cerebration. Any one of these criteria together with the overall picture of the patient serve as general indications for initiating dialysis. A deterioration in the clinical status is the single most important indication for dialysis.

Acute failure is commonly associated with surgical or other trauma, pancreatitis or peritonitis (or other forms of bacterial sepsis). It may also follow the administration of incompatible blood[46,47] or the ingestion of nephrotoxic agents such as ethylene glycol[24] or carbon titrachloride.[22,46]

Table 3

List Of Dialyzable Compounds Commonly Ingested

Barbiturate[7,36,37,9]	Boric Acid[45]
Salicylate[10,38,39,40]	Ethchlorvynol (Placidyl®)[26]
Glutethimide (Doriden®)[36,37]	Sulfa Drugs[35,46]
Methyl Alcohol[41,42]	Carbon Tetrachloride[22,46]
Ethyl Alcohol[41]	Bichloride of Mercury[35]
Phenothiazide[26]	Calcium[7]
Meprobamate[24]	Bromides[24]
Chloral Hydrate[26]	Diphenylhydantcin (Dilantin®)[24]
Sodium Chloride[43,44]	Potassium Thiocyanate[24]
Ethylene Glycol[24]	Ethyl-cyclohexyl Carbonate (Valmid®)[24]
Ethylene Dibromide[26]	
Mushroom[12]	

Chronic renal insufficiency. For patients with chronic kidney disease, dialysis may be indicated for patients who experience acute physiological insults which effect their renal function secondarily. Preoperatively, dialysis may be necessary to restore the patient's metabolic reserves when a favorable prognosis is anticipated. There are selected cases of advanced renal insufficiency that require peritoneal dialysis in anticipation of organ transplant or as candidates for a chronic dialysis program.[18,19,20,26,29]

Liver failure. Nienhuis, in a very well documented case, has defined the role of peritoneal dialysis in the treatment of hepatic coma.[28] Dialysis is especially indicated in cases of hepatitis associated with a deteriorating neurologic status, a rising blood ammonia and refractoriness to the usual medical therapy.[7,25,27,29] The prognosis is particularly poor when the total bilirubin exceeds 20 milligrams percent or if there is concomitant renal failure.

Exogenous intoxication. Peritoneal dialysis is indicated in the management of dialyzable poisons.[24,26,30] After securing an airway, supporting respiration and blood pressure, and retrieving all material possible by stomach lavage, the initiation of dialysis (peritoneal or hemodialysis) will shorten the comatose or toxic state and often prevent a fatility.[24] The clinician is subsequently guided by the serum levels of the poison and the progress of the clinical picture.[24] (See Table 3).

Intractable edema of cardiac origin. The literature contains cases of edema of cardiac origin refractory to the medical treatment. Some have been dramatically responsive to the use of hypertonic dialyzing solutions. These solutions are capable of removing an excess of 15 liters of water during 24 hours of dialysis.[7,21,28,33]

Endogenous intoxications. The syndromes of hyperuricemia with nephropathy secondary to the treatment of leukemia as well as hypercalcemia due to metastatic cancer, have, on occasion, been completely reversed by brief periods of peritoneal dialysis.[7,11,31,32]

Major fluid and electrolyte disturbances. The slow, steady rate of action in peritoneal dialysis renders the technique very effective in the gradual correction of imbalances in potassium, sodium, chloride and water.[7,21]

Toxemia of pregnancy. There are several very well documented cases of advanced toxemia of pregnancy complicated by congestive heart failure, convulsions and uremia refractory to the usual medical methods of treating this disease. These cases have responded quite favorably to peritoneal dialysis.[8,34,35]

Peritoneal Dialysis – Disadvantages of: Peritoneal dialysis is inadequate to meet the demands of an advanced metabolic disorder especially when associated with trauma or sepsis. The peritoneal approach may be inadequate in the face of combined hepatic and renal failure. The loss of proteins in peritoneal dialysis is a disadvantage though they may readily be replaced by plasma or albumin.[25] Peritoneal dialysis is only one-fourth as rapid as a Kolff twin coil kidney.[9,22,23,24]

The advantages of peritoneal dialysis in comparison with extracorporeal hemodialysis. Peritoneal dialysis by a more physiological rate of action causes less disturbance in homeostasis, a slower change in volumes and concentrations, lower incidence of cardiac arrhythmias and convulsions. Peritoneal dialysis is effective in the presence of circulatory collapse and is easily applicable to infants and children. The peritoneal approach does not expose the patient to the dangers of transfusion reactions, anticoagulation, septicemia, or air embolism. Current literature provides examples of successful dialyses performed in the face of fulminating infections (including infected wounds of the abdominal wall.[21,26] The peritoneal dialysis solutions provide an excellent source of calories by the absorption of 45 to 85 grams of glucose per lavage.[25] The anuric patient may enjoy an oral intake of food and fluids. Equipment for this procedure is simple. Less skill is required. Once the peritoneal catheter is in place, peritoneal dialysis may be performed by a nurse under a doctor's guidance. Minimal laboratory control is required for this procedure. It may be used in the patient's room.

Because of its relative simplicity and availability, peritoneal dialysis may be performed in the smaller community hospitals. It may be initiated early prior to and during a patient's transport to centers where hemodialysis is available.

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Office Evaluation of Streptococcal Infections of The Upper Respiratory Tract

Rowland B. French, M.D.

Although previous studies have been reported concerning the accuracy of clinical diagnoses of upper respiratory beta hemolytic streptococcal infections, it was thought that it might be of some value to evaluate this in a general office practice of patients complaining of a sore throat with or without the accompanying symptoms of cough, rhinitis or fever. Standard texts[1] describe streptococcal pharyngitis as being accompanied by some nasal discharge in 60% of patients, cough in one-half and hoarseness in some. Unfortunately, for diagnostic purposes, the pharynx may vary from marked inflammation to very slight; or largely consisting of lymphoid hyperplasia.

Diagnosis was made by fluorescent antibody method at the Diagnostic Laboratory of the Department of Health and Welfare, Augusta, Maine, and the following results reproduced by their kind permission.

One hundred and nine cases (almost consecutive) of patients complaining of sore throats were cultured by direct swabs, inserted into sterile glass containers and as soon as possible mailed to the Diagnostic Laboratory. Cases of herpangina, those that appeared to be simple rhinitis, or cases of bronchitis without sore throats were not cultured.

The study is also somewhat weighted toward positive streptococcal cultures as typical tonsillitis cases were included, and these usually turned out to be streptococcal in origin.

Of the one hundred and nine cases, 30 turned out to have positive cultures for beta hemolytic streptococcus. Each case was evaluated by making a notation at the time the culture was taken as to what the diagnosis was thought to be. In this study, 12 cases thought to give negative diagnoses turned out to be positive. This made up almost half of the positive cases. Twenty-two cases were thought to be positive and turned out to be negative. Sixteen cases were thought to be positive and were positive. Fifty-two were thought to be negative and were negative, making an accuracy of about 50% or less.

Diagnostically, if one is looking for a typical fiery red throat, one will miss many cases. Of the 30 positive cases, eight had no fever and in four others it was under 100 degrees. Five had coughs, largely non-productive. Six had some degree of rhinitis. In reviewing diagnostic errors, the presence of rhinitis and that of cough made one think more of a common cold. This occurred in the group thought negative and found positive as rhinitis and cough in two, cough only in one, and rhinitis only in three.

In the negative group, 32 had coughs, 27 had fever and 29 had rhinitis. In 11 a sore throat was present with no cough or rhinitis and yet not due to a streptococcus.

It was in this last group that the most errors occurred by calling cases positive that tests proved to be negative.

Doctor Okey, the Director of the Diagnostic Laboratory,[2] has written that the test is quite accurate in picking up positive cases; false negatives rarely occur; but there probably is some loss of viability in transit.

It is also of interest to note in his paper[3] there were numerous cases reported in a paper on streptococcal nasopharyngitis showing fever with no sore throat.

The importance of the accurate diagnosis of beta hemolytic streptococcal infections is the knowledge that rheumatic fever can be largely prevented by adequate treatment of the streptococcal infections. It would seem from this study and other similar ones, that clinical impressions of the existence of a streptococcal infection are inaccurate and that the fluorescent antibody method is of great help in determining adequate treatment.[4,5,6]

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Pathology of the Sjögren-Larsson Syndrome*

H. S. Baar, M.D., Ph.D., M.R.C.P., F.C. Path. and J. Galindo, M.D.

Sjögren and Larsson, in 1957, reported in the Scandinavian literature on a new familial syndrome characterized by congenital ichthyosis, oligophrenia, and spastic disorders which, occasionally, were accompanied by degenerative changes in the macula. Their study comprised 28 cases, 18 females and 10 males, all, with the exception of 3, from the county of Vasterbotten in the north of Sweden, an area where numerous consanguinous marriages occur.

A report on two siblings – natives of Maine whose family supposedly immigrated from Sweden 300 years ago – with this disorder, was published by Baar et al in 1960. In this paper, the literature was reviewed and the thesis put forward that the dermatosis is not a congenital ichthyosis, but an erythema ichthyosiforme secondary to a lesion of vegetative centers in the hypothalamus. In addition to the examples reviewed in that paper, one report was published by Richards et al, (1957), one by Schachter (1961), and one by Zaleski, (1962). Apart from the families studied by Sjögren and Larsson, only 6 examples of this disorder have been published, and none of the publications included a post mortem report. Meanwhile, one of the reported siblings died, and the present paper is the first report on the morbid anatomical findings in that disorder.

Case Report

E. H., female, born July 20, 1909. The clinical findings up to 1960 have been already reported (Baar et al, 1960). On June 18, 1960, her temperature rose to 101.6° and she started coughing. On June 24th she had twitchings of the right side of the face for the first time. That same day, twitching of the whole face was noted several times, each episode taking about one minute.

She was reported as unconscious during these attacks. The right pupil was dilated but reacted promptly to light stimulus. Dilantin® and phenobarbital were administered. The following day she had several convulsive seizures which were described as of the Jacksonian type. Between seizures, no pathological reflexes were elicited nor was papilledema or anisocoria present. A blood count taken on that day was: Hb. 12 g/100 ml.; RBC. 4,100,000; MCH 29; WBC. 13,350; polymorphonuclear segmented 85%; band forms 2%; lymphocytes 10%; monocytes 3%. The following day, the eighth before death, she had several convulsions which lasted from 15 to 20 minutes each. Her pulse was weak and the blood pressure was 76/40. Some râles were present on both sides. Sodium amytal was given to her and she had no further convulsions. The following day she was drowsy, the heart sounds were of good quality, but the rate was rapid. Both pupils were equally dilated. Since oral intake was virtually nil, she was given 2000 ml. of 5% glucose in saline, I.V. A few hours later, her blood pressure rose to 90/60 and she appeared somewhat better. Twenty-four hours later she was semicomatose but responded to painful stimuli. An additional two liters of I.V. 5% glucose in saline were given. Her general condition remained poor and, 5 days prior to death, twitching of the left corner of the mouth was noted.

Three days before death, the temperature rose to 105°F. NPN was 58 mg/100 ml.; Total Protein 6.3 g/100 ml.; Albumin 3.9; Globulin 2.4 g/100 ml.; Phosphorus 3.2 mg/100 ml.; Calcium 9.8 mg/100 ml.; Sodium 144 mEq/L; Cholesterol 185 mg./100 ml.; Cholesterol esters 140 mg./100 ml.; Alkaline phosphatase 3.18 Bodansky units and 9.8 King Armstrong units; Formol stable acid phosphatase;: 5 King Armstrong units; Thymol turbidity 2 units; Potassium 3.6 mEq/L; Urine slightly cloudy; pH 5.0; Specific gravity 1.022; Urobilinogen not increased, Porphobilinogen absent; Albumen 10 mg/100 ml.; Sugar negative; Acetone negative; Rare WBC; Few bacteria.

The following day, twitching was still present on the right side of the face; the temperature was 104°F. Heart sounds were clear but distant. Respiration was labored. She was continued on Dilantin and received again 1500 ml. of 5% glucose in saline I.V. The next morning, July 3, 1960, she had Chaine Stokes respiration, was agonal, and expired at noontime.

Autopsy Findings

At post mortem examination, the body was that of a small, greatly-emaciated, white female with pale skin that had a slightly yellowish tinge.

The skin of the entire body, with the exception of the face, was dry, glistening and showed a fine scaliness, which was lamellar on the extensor sides of legs and feet. The distribution of body hair was of a normal female type.

The knees and, to a lesser degree, the hips showed flexion contractures and there was a marked pitting edema of the legs and feet, noted also on the back.

The body length was 147 cm. and the stature proportioned. The head was 57.5 cm. in circumference, the mentobregmatic diameter 23 cm., and the bitemporal diameter 12.5 cm., foramen occipitale to glabella 15 cm.

The chest was asymmetrical, the right side bulging anteriorly more than the left. The circumference was 82.5 cm.

The internal jugular vein on the left side was conspicuous as a broad, firm cord, and was occluded by a blackish-red, moderately adherent thrombus that extended from the base of the skull to the clavicle, while the right jugular vein was entirely free in its lumen.

The thyroid gland was very small, 3.15 g. in weight, asymmetrical but normal in consistency. Microscopically, there was a slight variation in acinar size with a fair to good distribution of colloid without scalloping. The lining epithelium was cuboidal without infoldings and no crowding of acini was evident. The connective tissue was within normal limits.

The trachea contained some muco-purulent fluid and curdled milk. The pleural surfaces were smooth and lustrous and there were no adhesions or free fluid. The lungs showed numerous, scattered, pinpoint-sized areas of subpleural hemorrhage. Both lungs were pink, with the exception of the lower lobe of the right lung which was dark, blackish-red. The cut surfaces revealed abundant, pale yellow, frothy fluid and, microscopically, there was patchy bronchopneumonia.

The gastrointestinal tract was, for the most part, unremarkable save for some distention of the vessels around the cardia and the proximal third of the stomach. The liver was small, 770 g. in weight, brownish-red. It had a sharp anterior margin and, on the cut surface, the lobular pattern was distinct with the centers of the lobules depressed and dark red, surrounded

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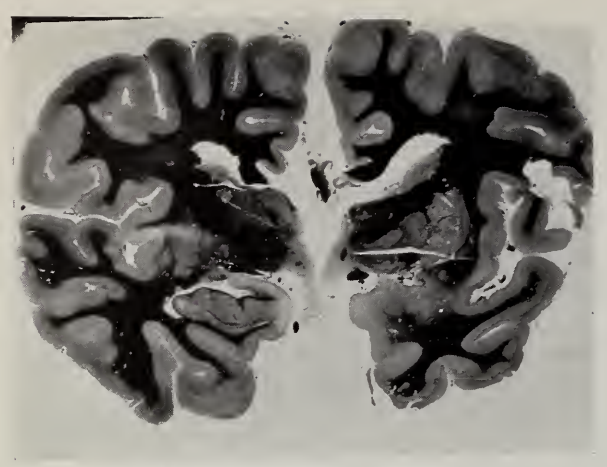


Fig. 1. Coronal section of brain in front of corpora mamillaria. Weil's myelin stain. Natural size. Note hypermyelinisation in right corpus striatum and atrophy of the head of right caudate nucleus.

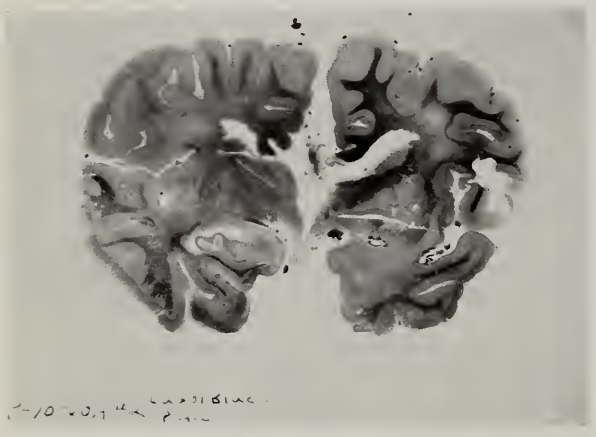


Fig. 2. Showing demyelinisation in centrum semiovale, mainly on the left side. Luxol Blue-P.A.S. Natural size.

by yellowish peripheries which corresponded, microscopically, to mild centro-lobular congestion and fatty metamorphosis.

The spleen was 80 g. in weight, soft, the pulp was diffluent, and the Malphigian corpuscles atrophic.

The kidneys were soft in consistency and weighed 85 g. each. Their capsules stripped off with ease. The external surface showed distended stellate venules and a few pinhead-sized spots. On the cut surface, the corticomedullary boundaries were scarcely discernible, in the periphery short, radiating red streaks were visible, while the rest of the kidney was pink-gray in color. Microscopically, the kidneys showed groups of hyalinised or fibrosed glomeruli and spherical calcium deposits in the Henle-loops, distal convoluted tubules and, occasionally, collecting tubules. The renal pelvis, the ureters, and the urinary bladder appeared normal.

The skull showed a mild basilar impression. Within the skull, a thrombosis was seen of the left sigmoid and transverse sinuses and the proximal part of the sinus rectus, which was continuous with the thrombus of the jugular vein.

The brain was 1300 g. in weight, showed depressed parietal areas of the cortex and a difference in size of the two hemispheres, the right one being smaller than the left. The most conspicuous finding was an asymmetrical atrophy of the heads of the caudate nuclei and a status marmoratus of the right corpus striatum. This is shown in Fig. 1. The actual dimensions of the heads of caudate nuclei in the coronal section in front of the mamillary bodies were 11 x 5 mm. on the right side



Fig. 3. Gliosis of cortico-medullary boundary. Holzer.

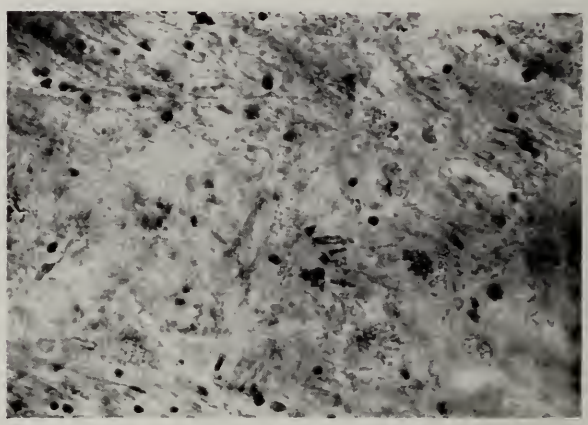


Fig. 4. Lipofuscin in lentiform nucleus. Celloidin.H.E. x 450.

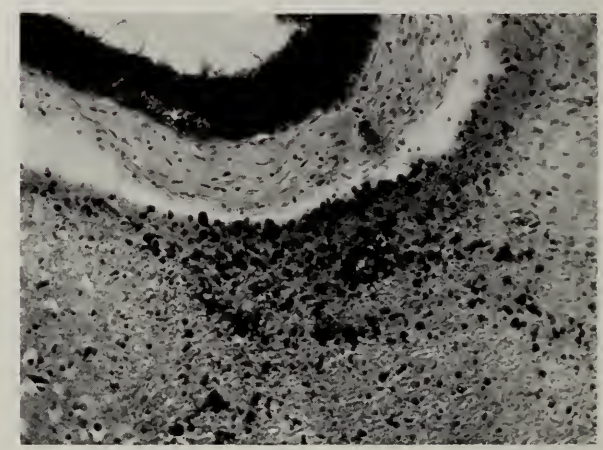


Fig. 5. Corpora amylacea in subpial cortex.H.E. x 150.

and 14 x 5 mm. on the left. Although the section is not exactly coronal, the difference was ascertained to be real and not apparent.

In addition to the hypermyelinisation in the corpus striatum, there were small patches of demyelinisation in the centrum semiovale. Fig. 2 shows such areas of demyelinisation in frontal lobes more marked on the left than on the right side. Marked gliosis was present in the whole central gray matter and in the cortical-subcortical boundaries, particularly in the frontal lobes (Fig. 3).

The number of neurons was decreased in the caudate and lentiform nuclei, particularly on the right side. Within the lentiform, as well in putamen and as in globus pallidus, the scanty nerve cells were separated by broad bands of myelinated fibres of an even width which, in places, were diffusely and



Fig. 6. Showing corpora amylacea and status spongiosus in subependymal area of third ventricle.H.E. x 150.

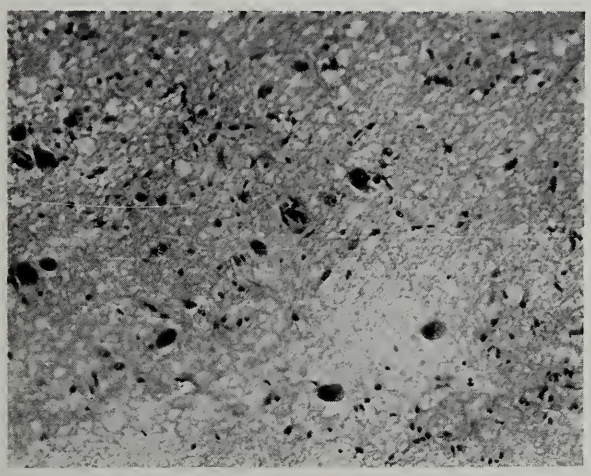


Fig. 7. Severe neuronal loss in right ventromedial nucleus.H.E. x 150.

irregularly tangled, and, in other places, occurred in the form of sharply demarcated islands. In addition to neuronal loss, there were ill-delineated areas of degeneration in the basal ganglia. These degenerative zones showed dense fibrillary masses of entangled glia with ballooning fibres, fragmentation of nuclei and disruption of axonal continuity. In many, scattered plump microglial elements with finely granular intracytoplasmatic lipofuscin were present (Fig. 4). While brown pigment granules were abundant, an occasional microglial cell was packed with dark purple granules. Between the glial fibres, and, in places, in the vicinity of vessels, a number of irregularly scattered, slit-like spaces was observed, characteristic of status cribrosus.

There were innumerable corpora amylacea which were stained basophilically with hematoxylin-eosin and showed the characteristic positive staining with P.A.S. Although seen in scattered locations within areas of neuronal loss and gliosis, the majority of such structures were collected in subpial regions, in the vicinity of larger blood vessels and in irregular festoons in the subependymal portion of the third ventricle (Figs. 5 and 6).

The ventricular system showed a moderate dilatation which was more marked on the right than on the left side. This mild hydrocephalus was obviously the result of shrinkage of gray matter.

Hypothalamic nuclei showed loss of neurones and gliosis.

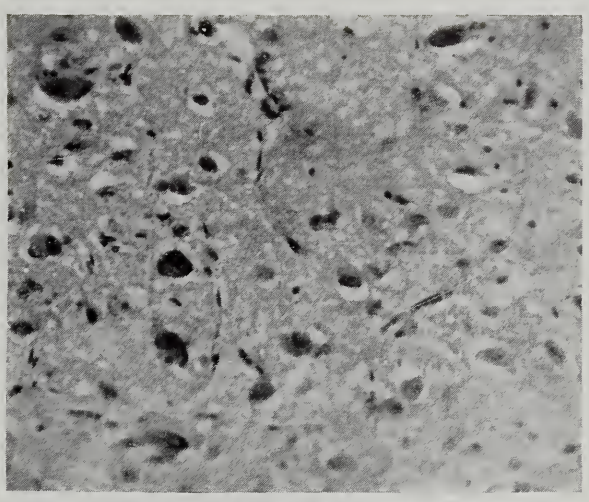


Fig. 8. Showing moderate neuronal loss and gliosis in left ventromedial nucleus.H.E. x 150.

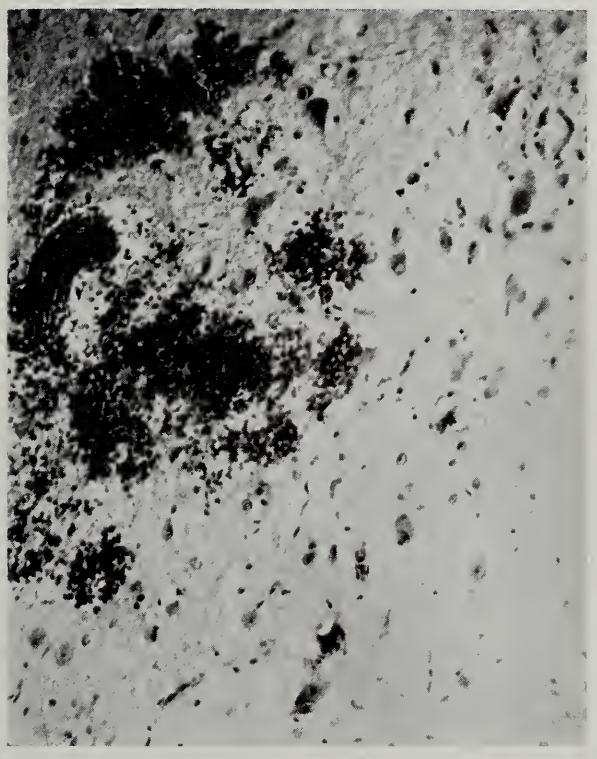


Fig. 9. Showing recent hemorrhage in right paraventricular nucleus.H.E. x 150.

The neuronal loss involved supraoptic, paraventricular, dorsomedial and ventromedial nuclei and was, in all, considerably more marked on the right side than on the left (Figs. 7 and 8). The whole sub- and hypothalamus showed spongy degeneration (Fig. 7). In addition to the chronic changes, there were small, recent, perivenous hemorrhages in the subependymal area of the third ventricle (Fig. 9).

The pathological process of recent occurrence, the result of jugular vein and intradural sinus thrombosis, was characterized by scattered microscopic hemorrhages in the lentiform nuclei, the internal capsule, and the subependymal areas around the third ventricle. These varied in dimensions containing a few red blood cells up to full-blown ball hemorrhages. Within the same areas there were irregularly-laced deposits of pigment which was brown in color, sometimes iron positive, but mostly iron negative. The brown, Prussian blue negative, pigment

stained bright red with Sudan IV. There were acute neuronal changes such as margination or disappearance of Nissl-substance. Occasionally, a focus of periventricular round cell infiltration was found. A few patches of neuronal loss in the fronto-parietal cortex were either the result of ischemia produced by the venous occlusion or secondary to the terminal epileptic fits.

The findings in the presented case are of interest for 3 reasons. First, the "paraplegia in flexion" mentioned in the clinical report (Baar et al, 1960). Our findings support the thesis of Yakovlev (1954) that this symptom is due, predominantly, to lesions of globus pallidus and frontal connections. Secondly, the case throws light on the origin of status marmoratus. The majority of cases are due to birth injury ("myelinated scars") (Schwartz 1924 and 1961), possibly thrombophlebitis in the Galenic system (Malamud, 1957). However, in the present case, it is, obviously, an integral part of a familial disorder and a malformation in nature, comparable with the familial cases of état marbré described by van Bogaert (1950 a and b). Finally, the changes in the hypothalamus support the hypothesis that the erythema ichthyosiforme is secondary to cerebral pathology, namely neuronal loss and gliosis of hypothalamic nuclei and their release from cortical inhibition.

Summary

The first report on anatomical changes in Sjögren-Larsson syndrome is presented. The basic changes were complicated by a terminal jugular vein and intradural sinus thrombosis. The relation of paraplegia in flexion to the findings in the presented case and the significance for the interpretation of status marmoratus are discussed. The hypothalamic lesions are considered as the basis for the erythema ichthyosiforme.

Acknowledgment

The authors wish to thank Mrs. E. Beverage for technical assistance and Mr. K. Allen for the photography.

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19th AMA Clinical Convention

Philadelphia, Pa. — Nov. 28-Dec. 1, 1965

A comprehensive scientific program, a new postgraduate course, and special clinical workshops are some of the features of the American Medical Association's 19th Clinical Convention, Nov. 28-Dec. 1, in Philadelphia.

An outstanding scientific program is designed to hold special interest for the practitioner. Some topics to be covered: ulcerative colitis, gram-negative bacterial infections, a medical-surgical review of cardiovascular surgery, drug therapy in rheumatology, and cancer chemotherapy and preventive surgery.

The practicing physician will be able to participate in one of the convention's new features. Clinical workshops on diabetes, examination of the heart, management of common eye problems, and the solution of selected diagnostic and therapeutic problems will be conducted by outstanding teachers.

Also new will be a postgraduate course in cardiovascular therapeutics. It will be offered in addition to the popular course on gynecology and obstetrics begun at the clinical convention last year in Miami.

The annual AMA conference on the Medical Aspects of Sports will be held the first day of the meeting, Nov. 28, in the Benjamin Franklin Hotel. It will be of special interest to high school and college team physicians.

There will be approximately 100 scientific exhibits, and 30 medical motion pictures.

Color television will be presented on the stage of the Civic Center in cooperation with the Hospital of the University of Pennsylvania. The subjects of six programs, to be followed by discussion, are "Lymphocytes, Cellular Immunities and Tissue Transplantation," "Renal Hypertension," "Pulmonary Resection," "Pulmonary Function Studies," "Surgical Aspects of Thyroid Diseases," and "Medical Aspects of Thyroid Diseases."

Twelve fireside conferences will be held Sunday evening, Nov. 28, at the Warwick Hotel. They will be joint sessions of the American College of Chest Physicians and the AMA.

W. Emory Burnett, M.D., is general chairman of the meeting. Donald A. Dupler, M.D., is chairman of the scientific program committee.

Ileostomy

Ignorance and Fear

Irving J. Poliner, M.D.*

Ulcerative colitis is often a severe, debilitating disease, the etiology of which remains unclear. Therapy, therefore, remains non-specific and supportive, including the use of adrenal steriod hormones. While there may be remissions, spontaneously or with therapy, there are frequent relapses. The only definitive and curative therapy is surgical, namely, total colectomy and ileostomy.

The physical and psychological problems of this procedure to a sick, debilitated patient seem immense. Recognizing their mutual problems, the first group of patients to have a colectomy and ileostomy at the New York City, Mt. Siani Hospital formed a "club." Most of these patients were on Ward Q or Ward T and thus the original name, the "QT Club." As more patients in different medical centers had this operation, more clubs were formed and consolidated into a nation-wide "Ileostomy Club." More recently, with the advent of ileo-bladders and numerous colostomies it has enlarged to become the "Ostomy Clubs."

Although the physician reassures a sick patient that an ileostomy (or ileo-bladder or colostomy) will function well, the objective evidence of seeing a "cured patient" who is "well" and leads a happy productive life is more convincing than any words. Conversely, by their desire to help patients with similar illnesses, the "ileostomists" are the most helpful associates the physician has. As pre- and postoperative "moral builders" to the patient, the ileostomists fullfil a great need. The physical, social, and sexual questions that patients have prior to the operation are much more fully discussed with an understanding ileostomist, who has gone through these procedures. Ileostomy is certainly no bar to the successful marriage or a successful pregnancy. Although personal hygiene may take a few more minutes, it is not difficult to maintain after the initial problems have been overcome. Many new improvements have been made in the appliances to the point where it is impossible to tell "who does and who doesn't have the ostomy."

Ileostomy organizations always have members who volunteer to do "missionary work." A physician may call upon these members preoperatively or postoperatively. They will not, however, visit a patient unless specifically requested by a physician. Frequently the nursing staff is not well trained in the management of an ileostomy or the appliance; and the physician, although better trained, may be too busy to give the patient the physical and psychological support necessary.

After discharge from the hospital the new "ostomist" may join a local ileostomy club. Through this organization he receives moral support during this difficult time. He is able to call on the experience of literally hundreds of ostomists while the physician himself may have experience with only one or two. The ostomy organizations themselves may call upon various physicians from within the community or outside of the community in regards to various aspects of their problems.

In 1964 a group of interested patients in Maine, New Hampshire, and Vermont formed the Northern New England Ileostomy Club. With the acceptance of colostomy patients, it became the Northern New England Ostomy Club. This organization has had wide and enthusiastic acceptance among ileostomy and colostomy patients proving the basic need and desire of these patients to take part in such a club.

Seven recent ileostomy patients in the Portland area and three young ileo-bladder patients have been helped by "old" members of this organization. Medical problems in northern New England are different from those at a large university center hospital. Hospitals are small, distances are great, and specialized medical information frequently is slow to arrive. For this reason the existance of this Northern New England Ostomy Club should be widely known to physicians, nurses, and the non-medical community. Many patients still "hide" their ileostomy from their friends and neighbors feeling that they are "abnormal." It is to aid these patients and future patients needing this type of operation that the organization enthusiastically offers its services.



Mrs. Adams

For further information or request for services contact Mrs. Marie Adams, President, 1236 Broadway, South Portland, Maine. Tel. 773-7328.

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Cryptococcosis
A Disease of Man and Animals

H. L. Chute,* F. L. Davis** and D. D. Payne†

Introduction

Cryptococcosis is a subacute or chronic disease of dogs, cattle, cats, horses, pigs and man caused by the fungus *Cryptococcus neoformans.*

The disease in dogs and cats is characterized by nervous and/or respiratory symptoms also subcutaneous granulomas are seen around the ears, face or feet. Mastitis is the common lesion in cattle. Respiratory signs and nasal discharge result from nasal granulomas in horses.

The disease in man in Europe has been reported to be characterized by skin lesions, whereas in the United States it has been primarily an infection of the central nervous system. In many cases where meningitis was the principal feature, lesions have been found in the lungs at autopsy and it is probable that in most cases the primary lesion was in the lungs.

Although there is no evidence of transmission of this yeast type fungus from animal to animal or from animal to man, contact with infected cats should be reduced to a minimum.

This paper covers the first known report of the disease in a cat from the State of Maine.

Literature Review.

The first case in England of cryptococcosis was reported from a cat in 1964.[1] The animal had a soft red mass extending throughout the right nasal fossa, and an ulcer below the right eye and at the upper commissure of the right nostril. There was a chronic nasal discharge. *Cryptococcus neoformans* was grown in culture, was seen in sections, and isolates were pathogenic for mice. Recently,[2] a case of cryptococcal meningitis was reported from a cat in North Queensland, Australia. This was reputed to be the first case in a domestic animal in that country. However, two cases have been reported in humans.

One of the first cases in cats in the U. S. was reported by Holzworth[3] in 1952 from the Boston, Massachusetts area. Since that time several cases have been reported from this country. One good review of the subject was reported in 1963.[4] Two cases were described in detail, one in which a cat with a subcutaneous facial lesion had recovered (by surgery) and the second being a blind cat with lung lesions as well as a meningitis. The organism was demonstrated in pigeon droppings taken from premises adjoining those of the owner of this blind cat.

A review[5] from Germany in 1963 stated that Cryptococcosis is usually in the upper respiratory and central nervous system. (CNS.) It occurs in the horse, cat, dog as a CNS disturbance; as a pulmonary infection in the horse, cat, dog, pig, mink; and as a mammary infection in the cow. Sources of infection can include soil, dust, milking machines, droppings of pigeons and other birds.

The disease has also been reported in swine, marmoset monkeys and a cheetah.

Although in *man* the disease is as yet rare, in recent years increasing numbers of cases have been reported. A detailed discussion of the disease in man by one author[6] states that up to 1936, of 56 cases on record, 49 involved the meninges and in 28 of these cases only the CNS was involved. It frequently takes the form of a chronic meningitis closely resembling tuberculous meningitis, or, it appears as an undiagnosable intracranial disorder, the signs and symptoms of which may resemble those of a brain tumor. Many of the cases are not diagnosed until autopsy, and all remain diagnostic problems until the specific organisms are demonstrated in the spinal fluid by culture or by especially careful microscopic study of sediment from the spinal fluid. The cutaneous lesions are lavender colored, papular nodules or acne-like eruptions that involve deep layers of the skin. Pulmonary Cryptococcosis presents the common clinical features of a widely disseminated, focal, chronic pneumonia.

Diagnosis.

Diagnosis is confirmed by identification of the organism, *Cryptococcus neoformans* from the lesions. The differential diagnosis is difficult and must not be confused with tubercular meningitis, cerebral tumor and abscess, lethargic encephalitis, dementia paralytica and lymphocytic choriomeningitis. This involves a mycological diagnosis by (1) identification of organism in the pus, spinal fluid or tissues, or culture of the pathogen. The organism will grow on acid dextrose Sabouraud's agar. In the tissues (histopathological) the organism has a wide mucoid capsule and displays prominent budding. It occurs in the tissues as an ovoid or spherical thick walled yeastlike body. The cell inside the capsule is usually 5 to 20 microns in diameter, the capsule overall is a maximum of 30 microns. The capsule stains selectively by the mucicarmine technique and the periodic

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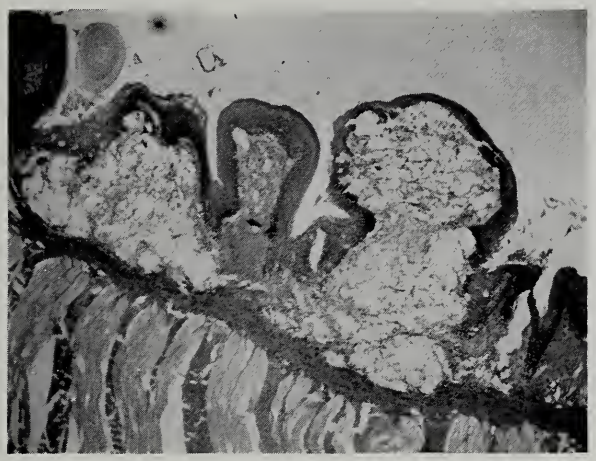


Fig. 1. Microscopic section of facial granuloma in a cat. The cyst-like lesion was filled with small round budding *Cryptococcus neoformans* organisms.

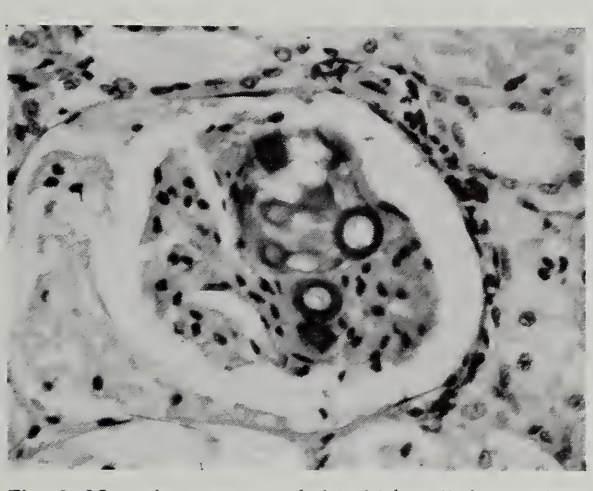


Fig. 2. Note the presence of the thick-walled organisms in the glomerulus of the cat kidney.

acid-Schiff (PAS)method for glycogen. (2) Animal inoculation. The easiest and quickest method of diagnosis is intracerebral inoculation of the mouse followed by death in 5-8 days, and demonstration of the organism.

Clinical History of Case.

During October, 1962 a feline patient was presented to a local veterinarian (F.L.D.) with tonsillitis. The animal responded well to penicillin, oral sulfas and vitamin therapy. In January, 1963, the patient had an acute febrile condition (Temp. 105.4°F.) It responded well to Chloromycetin® (Parke-Davis) therapy. In March, 1963 the cat was afflicted with sudden lethargy but with normal temperature. It responded again to Chloromycetin treatment. In February, 1964 the cat was presented with a severe sinusitis, drooling and sneezing but temperature normal. The cat was sent home and given nose drops consisting of Varizyme® (Lederle), Kymar® (Armour) and Biosol® (Upjohn) and oral administration of Aureomycin® (Cyanamid). On March 2 the owner presented the animal again in a debilitated condition and euthanasia was performed.

Pathology.

Around the lips, on the tongue and on the dorsal mucosa of the mouth, multiple granuloma-like lesions were prevalent. The mandibular and popliteal lymph nodes were enlarged. The kidneys were yellow and firm.

Histopathological Diagnosis.

The mucosal surface of the lips and tongue showed numerous large cyst-like formations. There was no inflammatory response in the surrounding tissue. The cysts contained connective tissue and numerous oval shaped globules which glistened like starch granules when stained with hematoxylin and eosin. (Fig. 1.) Mucicarmine stained tissue showed these granules to be Cryptococcus organisms, and budding frequently observed. The kidney showed extensive tubular degeneration and the organisms were noted to be lodged particularly in the glomeruli (Fig. 2.)

The *C. neoformans* appeared in the tissues examined as a round, single-budding, thick-walled yeast-like organism, 5 to 20 microns in diameter. The entire organism stained red with mucicarmine stain and in all respects had the characteristics of *C. neoformans.*

Summary

The clinical and pathological findings of the first case of *Cryptococcus neoformans* in a cat in Maine is described.

The ramifications of the disease in man and animals are described.

The prognosis is grave in man and animals, however, some favorable results have been shown with early treatment with amphotercin B.

In man the disease may be characterized by skin or central nervous system lesions. The lesions in man and animals show practically no inflammatory reaction. The organisms are widely scattered throughout the tissues and are round budding cells surrounded by a thick mucoid material which stains specifically with mucicarmine. The organism grows readily on acid dextrose (Sabouraud) agar.

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Use of Drugs in Pregnancy

E. Stanley Young, M.D.

Introduction

This is a review of papers by North American authors dating from 1961 through 1964. The papers reported on are of both clinical and research origins.

The subject of drugs used during pregnancy is of interest to all physicians, but more particularly to the general practitioners, obstetricians, and internists who see the bulk of pregnant women.

Considerable importance is given to the effect of drugs on the fetus by the Committee on Fetus and Newborn of the American Medical Association which has said, "when there have been no pharmacologic studies of a drug on immature subjects, an explicit statement of this fact should be indicated on the drug label or in a readily available package leaflet."[2] This is also the policy of the Food and Drug Administration in licensing new drugs.

Whenever potentially toxic drugs are to be used in women who may have an early pregnancy, it may be wise to attempt diagnosis of the pregnancy by immunologic tests such as Pregnosticon (Organon), U.C.G. Test (Wampole), or Gravindex (Ortho).[5] Such tests can be completed in about two hours, and are fairly reliable as early as four weeks gestation.

Central Nervous System Depressants

(A) Anesthetics. All anesthetic drugs given to the mother are depressing to the fetus to some extent. Nitrous oxide, properly given, is probably least depressing to the fetus.[6] Ether is quite depressing, but is rapidly excreted in the infant who is ventilating. Trilene is found in infants blood in higher concentration than in the mothers blood; however, the fetus is seldom depressed, except when the drug is used in high dosage over a long period of time; when Trilene is self-administered by the mother, depression of the newborn is not a problem. Halothane and Cyclopropane are more depressing than ether. Curare and succinylcholine apparently do not enter the fetal circulation to any extent.[6]

(B) Hypnotics and Sedatives. In experimental animals Nembutal® given intravenously to the mother caused a deterioration of Electrocardiogram in the fetus.[3] However, in clinical experience, barbiturates, chloral hydrate, and ethanol cause significantly less depression of the newborn than the mother. Paraldehyde, on the other hand, is quite depressing. Tranquilizers, in general, do not cause much newborn depression, and Phenergan® specifically is quite safe in this regard.[6] Dramamine®, Cyclizine, and Meclizine are considered safe, in that no teratogenic effects have been noted in their long term use for nausea in pregnancy.[6,8] Thalidomide® causes phocomelia, hearing loss and fetal death.[8]

(C) Analgesics and Antipyretics. Narcotics cause distinct respiratory depression in the infant at delivery. This is true of Nalline® and Lorfan® as well as other narcotics.[4] Even as little as 50 mg of Demerol® can cause significant depression.[6]

Narcotic addiction in the mother is a cause of small size and withdrawal syndrome in the infant. The infant may require sedation for hyperirritability, vomiting and convulsions; the death rate is high in these infants.[4]

Salicylates have been reported to cause hemorrhage in the newborn when used in large doses near term, apparently due to hypoprothrombinemia. It has also been reported that ingestion of 15-18 grams of Acetyl Salicylic Acid by a mother 27 hours prior to delivery caused obvious salicytate poisoning in the newborn.[1] In this case the child did well; blood salicylate fell from 35 mg % to 22 mg % following an exchange transfusion.

(D) Anticonvulsants. There is very little information on this point, although several of these drugs are known to cause damage to the liver and bone marrow.

(E) Reserpine. This drug is mentioned specifically because it can cause serious nasal congestion and respiratory distress in the neonate and even death, if treatment is not given promptly in the form of an oral or nasopharyngeal airway.[4]

Local Anesthetics

Very little is known about the effects on the fetus of these drugs given to the mother. There is one case report of inadvertant administration of procaine to a fetus during attempted pudendal block. This infant showed central nervous system depression and shock, but survived with supportive treatment.

Autonomic Drugs

Parasympatholytic drugs cause a mild tachycardia in the fetus.[6] In experimental animals, it could not be shown that epinephrine crossed the "placental barrier" when injected intramuscularly in the mother.[3] However, uterine artery flow was decreased and fetal arterial pressure decreased. It is interesting in this regard that pheochromocytoma in the mother is very hazardous for the fetus. In one series of 24 cases the fetal mortality was 58%. Early surgical intervention is suggested in these cases.[3]

Diuretics

Rare cases of thrombocytopenia have been reported, apparently due to thiazide derivaties.[8]

Oxytocics

Oxytocics may cause fetal damage if overdosage occurs. This may happen in one of two ways – fetal as-

phixia may occur due to tetanic contraction of the uterus or head injury may occur due to tumultous labor.

Antimicrobial Drugs

Antibiotics theoretically may injure the fetus due to many different toxic effects. However, documented case reports are quite rare.

Two instances of fetal effects are fairly well demonstrated; tetracyclines cause discoloration of the teeth in the fetus;[4] streptomycin has caused hightone deafness in the fetus when used in long-term therapy of the mother.[7] Only about six such cases have been reported and streptomycin must be considered as a safe antibiotic to use for short-term therapy.

It is probable that Chloramphenicol has caused instances of hepatic damage in the fetus, similar to that seen in overdosage in the newborn or premature; case reports are few.

Sulfonamides have been known to cause hemolysis leading in hyperbilirubinemia, sometimes requiring exchange transfusion; again, this is a rare occurrence.[4,5] A great many antibiotics can be considered potentially toxic to the fetus; known to be hepatotoxic are Chloramphenicol, Chlortetracycline, perhaps Novobiocin®; known to be nephrotoxic are Bacitracin, Polymyxin B, Neomycin, Kanamycin, Vancomycin, and Ristocetin®.[4]

Hormones and Endocrine Drugs

Progesterone itself is not masculinizing. Most of the synthetic progestins can cause masculinization of the female fetus and advanced bone age. Duphaston® is least apt to have this effect and Provera® is seldom implicated. All of the androgens, and also diethylstilbestrol in large doses are masculinizing.[4]

Adrenal corticoids and ACTH have been shown in animals to produce abortion, cleft palate, undersized newborns, and high mortality in the newborn.[4] These effects have not been proven in humans. Fetal adrenal suppression has not been proven. Rules of clinical use are suggested:

(1) These drugs should be used only with trepidation in the first trimester.

(2) The smallest effective dose should be used.

Drugs used in treating hyperthyroidism have caused fetal effects. Iodine may cause goiter. Radioactive iodine can cause cretinism in the fetus. Thiouracils may cause goiter with increased, normal, or decreased thyroid function.[4]

Antimetabolites

Most anticancer drugs have been shown to be teratogenic in experimental animals. The only one known to be teratogenic in humans is aminopterin when used in the first trimester.[4] This drug as well as Methotrexate® has caused abortions when used in the first trimester. Methotrexate has been used in attempts at criminal abortion, many of these successful but injurious to the mother.

Miscellaneous

Smallpox vaccination in the mother has caused fetal vaccinia.[5] Influenza vaccination has been shown to cause increased anti – A and anti – B titers in the maternal circulation; it is not known whether this is of clinical significance.

Digitalis crosses the placenta readily but is not known to be toxic to the fetus.

Hypervitaminosis A has been associated with cleft palate in the infant.[8]

Summary

Although a great many drugs are known to be potentially toxic, only a few have been shown to cause serious fetal damage. The drugs most commonly used in routine prenatal care, namely thiazide derivatives and drugs in the cyclizine-meclizine class, can be considered very safe.

There are three general areas in which caution must be used.

The first of these is in the use of these drugs in the first trimester: ACTH, Corticosteroids, Androgens, masculinizing progestins, and Stilbestrol.

The second area is in the use of antimetabolites and antithyroid drugs at any time during pregnancy. Hyperthyroidism in pregnancy, when it is mild, can often be treated symptomatically with barbiturates or tranquilizers. When treatment is mandatory, the safest approach is a brief course of iodine and a thiouracil drug, followed by thyroidectomy.

The third area of concern is in the use of depressing drugs during labor and delivery. In this regard it should be pointed out that narcotics are relatively more depressing to the fetus than either barbiturates or tranquilizers.

In short, the physician must always remember that he is treating two patients rather than one.

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Poland Spring, Maine

Emergency Treatment of Pierre Robin's Syndrome

WILLIAM W. WARD, M.D. and EMERY B. HOWARD, JR., M.D.

Pierre Robin's syndrome is a congenital defect of the palate and hypoplasia of the mandible. Posterior displacement of the tongue causes life-threatening respiratory obstruction from birth. The following case is presented to emphasize an easy method to immediately relieve this respiratory obstruction.

The mother's pregnancy was complicated only by marked polyhydramnios. Forceps were used for delivery. At birth the child had Pierre Robin's syndrome with respiratory obstruction and an imperforate anus. An x-ray of chest and abdomen showed air below the diaphragm only in the stomach. Soon after birth the child was taken to the operating room. Without anesthesia a heavy silk suture was placed through the posterior tongue. The ends of the suture were passed through the anterior lower alveolar ridge and tied. Immediate and permanent relief of all respiratory obstruction was obtained. Abdominal exploration revealed duodenal atresia, malrotation with duodenal bands, many other bands between multiple loops of bowel, Meckle's diverticulum, hydronephrosis and hydroureter on the right. Lysis of adhesions, gastro-jejunostomy, excision of appendix and Meckle's diverticulum, gastrostomy and colostomy were performed. Postoperatively, although the airway remained clear, the child's color continued poor, out of oxygen, and he died eight hours after surgery.

In addition to the operative findings, autopsy revealed a large ventricular septal defect, hydronephrosis and hydroureter on the left and bladder outlet obstruction from congenital urethral valves. The head was not examined.

The child with Pierre Robin's syndrome is a constant nursing problem. Methods to relieve the respiratory obstruction have included tracheostomy, suture of the anterior tongue to lower lip and placement of a Kirschner wire through the tongue and mandible. Tracheostomy in the newborn is not without complications and is a nursing problem. The tongue and lower lip suture often pulls out in a short time.

In 1960 four newborns from the Boston City Hospital[1] with Pierre Robin's syndrome were treated successfully with a suture passed through the posterior tongue and anterior lower alveolar ridge. The one child of this series who was followed for a considerable length of time did not develop any disturbance of the growth of his teeth.

SUMMARY

A child with Pierre Robin's syndrome and other anomalies is presented. An immediate, effective and easy treatment for the respiratory distress is discussed.

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Special Article

The Medical School Of Maine

*Remarks by President James S. Coles**

We are met to commemorate the Medical School of Maine, an institution intimately associated with Bowdoin College for a full century of service, and to recognize especially this building, Seth Adams Hall, which was for more than half of that century the center of activity of that School.

It is customary, on such occasions, to recognize an anniversary of some sort, preferably a centennial, or at least, the passage of some period of years divisible by twenty-five, if not by fifty. This we cannot do today, with reference to the School or to the Building, but we can pay tribute to Doctor Alfred Mitchell, Dean of the School and President of the Maine Medical Association, who received his M.D. at Columbia a hundred years ago and who was a physician and teacher of physicians in this town and in this building for fifty years, from 1865 until his death in 1915.

Soon after William Allen accepted the presidency of Bowdoin in 1819, he wrote to Dr. Nathan Smith, the founder of the Dartmouth Medical School, then professor of the theory and practice of medicine at Yale, about the need for medical instruction in the new State of Maine. Dr. Smith encouraged him to pursue the project, and expressed the hope that State aid might be forthcoming. This hope was realized, for "the first legislature of Maine, to its honor and to the material increase of the health and happiness of the people" — here I quote the late George T. Little — "established, June 27, 1820, the Medical School of Maine, to be under the control of the Trustees and Overseers of Bowdoin College," granted fifteen hundred dollars for books, apparatus, etc., and authorized an annual payment of a thousand dollars.

Instruction began in the Spring of 1821, in what were supposed to be temporary quarters in Massachusetts Hall. As Mr. Little puts it, "Time moved on but the school did not. The building came to be spoken of as the Medical College."

In 1861, Adams Hall was built, following designs prepared by the Secretary of the Medical Faculty, Professor Paul Chadbourne, later President of Williams College. Speaking at the dedication of the building, in February, 1862, President Leonard Woods acknowledged indebtedness "to the State, for the grant of half a township of land," to the Maine Medical Association, for "friendly cooperation," to the College, for a contribution of $5,500 matching that of the State, and "to the liberality of a gentleman in Boston," now known to have been Seth Adams.

The Medical School closed its doors in 1921, primarily because its financial situation was such that it could not qualify as a "Class A" school. Closing had also been recommended in Dr. Abraham Flexner's authoritative report on "Medical Education in the United States and Canada," published in 1910 by the Carnegie Foundation for the Advancement of Teaching. In his report of that year President Sills wrote "For those who have known the College intimately, the closing of the Medical School naturally brings great regret. The College has had so many points of contact with the medical profession of Maine throughout these hundred years, and has received so much generous and loyal support from graduates of the Medical School that the abandonment of the school means a very great loss. The loss, however, is as nothing compared with the impairment of reputation, both of the school and the college, which would have followed the inability to maintain the school properly. The School closes its century of existence with a creditable record and in good standing."

Over the years, well over two thousand men received their M.D. degrees from the Medical School of Maine. More than half as many more were enrolled as students for a term or more. Of these, hundreds went on to complete their work elsewhere, and to become practicing physicians.

Though the Medical School is no more, Bowdoin College is still sending men into Medicine. Fifty or more Bowdoin graduates have been enrolled in one Medical School or another in the year just closed, and some 15 of the 170 men who will be graduated here tomorrow have been accepted for admission to a Medical School in the fall. The Garcelon and Merritt Fund, established in memory of two graduates of the Medical School, is each year helping Bowdoin men and men from Maine to complete their medical education.

It is good for us to pause at this 160th Commencement for a moment of recognition of the ties that have bound the College to the medical profession, and to mark this old building as the seat and center of a proud and honorable institution, the Medical School of Maine.

*Delivered at the 160th Commencement of Bowdoin College, June 11, 1965.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Current Items From The Diagnostic Laboratory

CHARLES H. OKEY, Ph.D.*

PHENYLKETONURIA DETECTION

The Department was authorized by an act of the recent legislative session to require hospitals, maternity homes and other maternity services to test or cause to be tested newborn infants for the presence of metabolic abnormalities which may be expected to result in subsequent mental deficiencies. The only exception to the requirement is in the case of a child whose parents object on the basis that such test conflicts with their religious tenets and practices. Rules and regulations defining this act have been distributed to the appropriate institutions under the program carried out jointly by the Division of Maternal and Child Health and the Diagnostic Laboratory.

Phenylketonuria is the only condition for which testing is required at the present time. Two-part blood collection kits have been provided to the institutions involved. Part one of the kit is to be used in the hospitals for procuring a blood specimen suitable for a phenylalanine determination by the Guthrie test performed in the Diagnostic Laboratory. Part two is to be given to the mother by the hospital with instructions to arrange for a follow-up specimen four to six weeks later. This specimen also will be tested in the Diagnostic Laboratory. The results of initial testing will be reported to the respective institutions. Follow-up specimen results will not be reported except in the case of a positive presumptive result which requires further testing. In the latter case, additional blood specimens will be tested with a quantitative chemical procedure and by chromatographic analysis. The Maternal and Child Health division will provide consultative service in cases of proven phenylketonuria.

Two presumptive positive phenylketonuria infants were screened in the detection program during early September and were in the process of being confirmed as this was being written. If confirmation is obtained, these infants will be the first cases found since testing began in September, 1962. Earlier testing, up to December, 1964, during which 10,000 infants were screened, was carried out as part of a national evaluation of the Guthrie bacterial inhibition assay method of detecting infants with excess blood phenylalanine. Thirty-nine confirmed cases of PKU were found in the 400,000 infants tested nationally. This number was approximately double the incidence expected on the basis of previous estimates. At the conclusion of the evaluation series a PKU detection program was developed jointly by the Division of Maternal and Child Health and the Diagnostic Laboratory to offer screening tests to all hospitals who wished to submit specimens. Confirmatory testing was made available through the facilities at Pineland Hospital and Training Center. Approximately 25,000 infants were tested without finding a single confirmed case.

THROAT CULTURES

A new throat culture swab outfit is being distributed by the Diagnostic Laboratory. The outfit is designed to transport and maintain viability for Group A beta hemolytic streptococci but is not entirely satisfactory for other uses. The swab itself is made of dacron which has the advantage of being free of inhibitory fatty acids normally found in untreated cotton swabs. The mailing envelope offers the advantages of reduced postage and ease of handling and storage over the older cardboard mailer and glass tube.

The American Heart Association and its state component, the Maine Heart Association recommend the use of throat cultures as part of the effort toward rheumatic fever prevention. The Associations point out that carefully controlled studies show clearly the lack of correlation between clinical signs and symptoms and the demonstrated presence of Group A streptococci. The severity of symptoms was not a reliable index in that viral infections were shown to produce severe reactions and mild disease was observed in the presence of streptococci. Cultures are considered the best basis for judgment as to the presence of streptococcal disease due to the Group A organisms.

In using the dacron swab, a three to five minute dry-

*Director, Diagnostic Laboratory

ing period should elapse before returning the swab to the envelope.

Gonococcus Culture

Culture service for gonorrhea diagnosis is available now to physicians of the state. The service is made possible by the use of a transport medium which has been shown to be an acceptable material for maintaining the viability of the gonococcus. Essentially, the medium is a non-nutritive anaerobic gel that maintains the status quo of the sample in terms of numbers of organisms. Some reservation must be expressed in that the transport medium is not totally efficient but the advantage it has over the smear is sufficient to recommend its use.

Many studies have shown the cultural method of diagnosis to be superior to the smear method by a factor of two to three-fold. A smear is considered adequate for the acute attack in the male, marginally adequate in the acute phase for the female and totally inadequate in the chronic stage of the disease in the female. Cultures are particularly useful in establishing an asymptomatic carrier state in females named as contacts and found negative by smear.

Close attention must be given to the instructions accompanying the gonorrhea culture outfit if success is to be expected using the transport medium. Adequate mechanical cleansing of the area to be sampled followed by an effort to express exudate from deeper areas will yield a better specimen than a superficial swipe of an undisturbed area. It is entirely practical to culture the rectum if this site is suspect. A culture medium of selective inhibitory quality is used which prevents the normal rectal flora from covering the culture surface yet allows luxuriant growth of the gonoccocus to take place.

Febrile Agglutinations

Each week, the Diagnostic Laboratory receives a number of blood specimens with accompanying requests for typhoid and paratyphoid agglutination tests. During the past four years, there has not been a single instance of a diagnostic titer reported except when the patient's diagnosis had already been established on cultural grounds. There have been a number of instances in which a great deal of excitement and confusion stemmed from an elevated titer which was eventually traced to recent typhoid immunization.

Antibody responses in typhoid and paratyphoid fevers occur in from one to two weeks after the onset of symptoms. Furthermore, the titers may require some additional time to reach diagnostic levels. For all of these reasons, it is recommended strongly that suspicions of typhoid or paratyphoid fever be allayed with stool cultures rather than agglutination tests.

SPECIAL COMMITTEES 1965-1966

Special Committees for 1965-1966 as appointed by the President, John F. Dougherty, M.D. of Bath.

(*Continued from September issue – page* 207)

*Medical School For Maine Committee

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*Appointed by the Council of the M.M.A. on 3/7/65
**Appointed by the Council of the M.M.A. on 9/12/65

Maine Heart Association Notes

116 State Street, Augusta, Maine

Diagnosis and Treatment of Digitalis Toxicity

"Clearly, although the gastrointestinal symptoms and cardiac irregularities are common findings, less frequent evidences may be more striking and therefore most important in diagnosis. Some of these—for example, green or yellow vision—are not uncovered unless the physician inquires about them. Prominent as evidence of digitalis toxicity in recent years is an increasing severity of congestive heart failure when the patient is on adequate or fairly large doses and the clinical picture progresses without apparent cause. Unexplained cerebral symptoms, dizziness, syncope, evidence of toxic psychosis, paresthesia around the lips and hands and trigeminal neuralgia fall into this category. Some of the usual indications for digitalis, such as increasing ventricular rates without alteration in the pacemaker mechanism in atrial fibrillation, may develop as the result of the drug. . . . Sudden tachycardia or bradycardia, changes from regularity to irregularity of the heart or the reverse occurring during therapy of congestive heart failure may be manifestations of digitalis toxicity.

". . . Constant observation by the physician and by the patient. therefore, are vital to the prevention and recognition of digitalis toxicity and are consequently primary to treatment."

Submitted by Jacob B. Dana, M.D.

Reference: Sodeman, W.A., M.D., N.E. Journal of Medicine 273:35-37, 1965

LOMOTIL—*Pharmacologic Activity.*

The significant pharmacologic actions of Lomotil are summarized as follows:

Evidence indicates that Lomotil acts directly by inhibiting excess peristalsis.

Lomotil is not known to inhibit nonpropulsive intestinal movements.

Roentgenograms demonstrate that this activity occurs within two hours after oral administration and persists for at least six hours.

Studies in the rat show Lomotil to be more effective in inhibiting fecal excretion than either codeine or morphine.

Analgesic, anticholinergic, mydriatic and gastric secretory effects have not been significant.

Reduction of propulsive motility with Lomotil relieves spasm and cramping, allows physiologic absorption of fluid and reduces frequency of evacuations to provide prompt, symptomatic control of virtually all diarrheas.

LOMOTIL® tablets liquid

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride .....2.5 mg.
(Warning: May be habit forming)
atropine sulfate .....0.025 mg.

slows propulsion · relieves distress · stops diarrhea



Precautions: Lomotil is an exempt narcotic preparation of very low addictive potential: more than three million prescriptions have now been written for Lomotil. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

Dosage: For full therapeutic effect – Rx full therapeutic dosage. The recommended initial daily dosages, *given in divided doses*, until diarrhea is controlled, are:

Children:
3 to 6 months–3 mg. (½ tsp.* t.i.d.)
6 to 12 months–4 mg. (½ tsp. q.i.d.)
1 to 2 years–5 mg. (½ tsp. 5 times daily)
2 to 5 years–6 mg. (1 tsp. t.i.d.)
5 to 8 years–8 mg. (1 tsp. q.i.d.)
8 to 12 years–10 mg. (1 tsp. 5 times daily)

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) **Based on 4 cc. per teaspoonful.*

Maintenance dosage may be as low as one fourth the therapeutic dose.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdosage.

SEARLE *Research in the Service of Medicine*

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

CANCER OF THE HEAD AND NECK

Edwin C. Pound, Jr., M.D., *Atlanta*

Perhaps no other area of the body, comparable in size, gives rise to as many anatomic varieties of malignant tumors as the head and neck. The great majority of tumors arising in this area are readily accessible and should undergo early diagnosis and treatment; however, cases are often seen in which a state of incurability has been reached due to a patient's own neglect from fear or incredibility of his disease state or delay in the institution of proper diagnostic and therapeutic measures by more responsible parties.

Treatment Important

Treatment of the primary lesion is of greatest importance and requires detailed knowledge of the natural history and characteristics of each tumor. With the exception of melanomas and thyroid carcinomas, which have a high incidence of vascular metastasis, most neoplasms of the head and neck spread mainly through lymphatic channels, which are entered by direct invasion. This spread is usually unilateral but can be bilateral if the primary tumor is located near the midline or if enough blockage occurs on one side of the neck to cause retrograde leakage to the other side.

Death from cancer arising in the head and neck area usually occurs with the primary lesion and metastases still confined above the clavicles. Therefore, operative procedures have been devised which widely remove the primary tumor in conjunction with the pathway of lymphatic spread. Although these operations may be quite radical in scope with wide sacrifice of tissue, present day reconstructive procedures can be utilized, often primarily, to make the end result a satisfactory one both cosmetically and functionally.

Bilateral cervical metastases should not be considered a contraindication to surgery since the five-year cure rate for these unfortunate individuals is approximately 30 per cent. Distant metastases, fixation of cervical nodes to underlying structures, evidence of secondary lymphatic skin invasion, and severe physical disabilities, however, are contraindications except to palliative procedures.

The merits of irradiation versus surgery in the treatment of head and neck cancer have often been expounded by advocates of one school or the other, but it is generally accepted that both forms of treatment have a place and depending upon the clinical situation, each can be used alone or in combination with the other for the best end results.

Chemotherapy

In recent years a new phase of cancer therapy has been established in which attempts have been made to control or destroy the growth of certain forms of cancer by chemotherapeutic techniques. Isolated perfusion or intra-arterial infusions now allow cytotoxic compounds to reach areas of neoplasia in amounts greater than previously possible by systemic methods. These compounds are particularly injurious to cells which divide frequently, such as cancer, but toxicity to the normal bone marrow still limits the amount which may be used. Extensive research is now underway for analogues of these compounds which are more selective for cancer cells.

Relief of pain is usually striking and at least some temporary regression in growth occurs following most regional perfusions with these compounds; therefore, these techniques have become a valuable adjunct to the therapy of head and neck tumors.

Summary

Earlier diagnosis and modern therapeutic techniques offer longer survival with less disfigurement and pain than ever before for unfortunate patients with head and neck cancer.

1293 Peachtree Street, N.E.

Approved by Professional Education Committee, Georgia Division ACS.

Reprinted from The Journal of the Medical Association of Georgia, April, 1964, Vol. 53, Page 134.

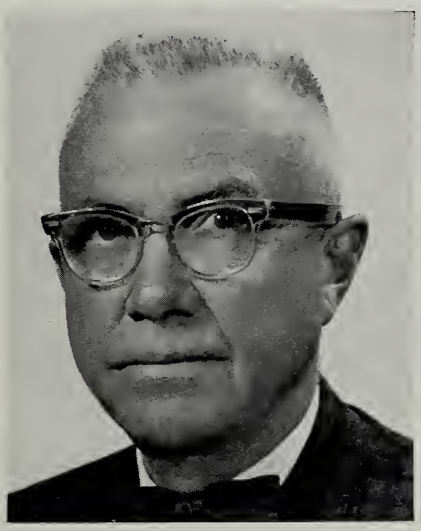
Necrologies

Ralph C. Stuart, M.D.
1896-1965

Dr. Ralph Stuart died September 18, 1965 after a short illness.

He was born in Calais, Maine in 1896. A graduate of Georgetown University he received his medical degree from the School of Medicine in 1921.

Except for a short time in practice at Calais his entire medical career was spent in Sangerville and Guilford.



He was a tireless worker for the profession, having served on most major committees of the Maine Medical Association. He was Councilor from the Sixth District, Chairman of the Council in 1962 and became President in 1963.

One of the last of the pioneer surgeons in the area, ever-kindly and professionally correct, he was friend and family physician to a large portion of Piscataquis County for over forty years.

"The skill of the physician shall lift up his head—
In the sight of great men he shall be praised."

Linus J. Stitham, M.D.

Roland L. McKay, M.D.
1884-1965

Roland L. McKay, M.D., 80, of Augusta, Maine died on May 24, 1965. He was born in Vassalboro, Maine on September 8, 1884, son of Greenwood H. and Ida Austin McKay.

Dr. McKay graduated from Brunswick High School, attended Bowdoin College and received his medical degree from Bowdoin Medical School in 1908. He interned at the St. Barnabas Hospital, the Augusta State Hospital and began his general practice in Augusta in 1909. During World War I he served as a Captain in the Medical Corps.

Dr. McKay was an Honorary member of the Kennebec County Medical Association and the Maine Medical Association, having received a 50-year pin in 1958 and a 55-year pin in 1963. He served as Councilor for the Fifth District of the Maine Medical Association from 1945 to 1947. He was also a member of the American Medical Association, a member of the staff of the Augusta General Hospital 31 years and chief of staff 10 years, Kennebec County Medical Examiner from 1924 to 1949, president of the Maine Medico-Legal Society in 1944 and had served as chairman of the State of Maine Board of Registration of Medicine.

Dr. McKay was a member of the Selective Service Appeals Board from 1941 to 1943 and 1951 to 1955, chairman of procurement and assignment of doctors from 1943 to 1946, a member of the War Manpower Commission, medical examining officer of the National Guard in 1942 and a member of Masonic bodies including Kora Temple Shrine.

Surviving are two sons, Murray H. of Dunbarton, New Hampshire and Roland A. of Phoenix, Arizona; seven grandchildren and six great-grandchildren.

Raymond E. Weymouth, M.D.
1903-1965

Raymond E. Weymouth, M.D., 62, of Bar Harbor, Maine died on August 20, 1965. He was born in Millinocket, Maine on January 6, 1903, son of Frank D. and Lottie M. H. Weymouth.

Dr. Weymouth graduated from Higgins Classical Institute in 1920, Colby College in 1925 and received his medical degree from Jefferson Medical College in 1932. He taught school in Houlton, Mattawamkeag and Bar Harbor from 1926 to 1928, was a member of the State Police Force summers from 1925 to 1930 and practiced medicine in Bar Harbor from 1933 until his death.



He was a member of the Hancock County Medical Society, the Maine Medical Association, serving as Councilor for the Fifth District for three terms from 1944 to 1952, 1955 to 1958 and 1961 to 1964, a member of the American Medical Association, the Alpha Omega Alpha Medical Honorary Society at Jefferson Medical College, the Academy of International Medicine and was a fellow of the American College of Physicians. He was also a member of Anah Temple, Masonic Lodge and Odd Fellows and had served in the Army Air Force Medical Corps from September 1942 to November 1945 holding the rank of Major. Dr. Weymouth had served as chief of staff of the Mt. Desert Island Hospital for 18 years.

Surviving are his widow, Alma W. Weymouth of Bar Harbor; a son, Raymond E., Jr. of Bar Harbor; two daughters, Mrs. Carolyn Drew of Bowie, Maryland and Mrs. Barbara Tarryk of Long Beach, California; his stepmother, Mrs. Frank Weymouth of Brewer and a stepbrother, John Dority of Brewer.

G. E. C. Logan, M.D.
1905-1965

G. E. C. Logan, M.D., 59, of Portland, Maine died on June 6, 1965. He was born in Maple, Ontario, Canada on September 17, 1905, son of Dr. and Mrs. Harris Logan.

Dr. Logan received his medical degree from the University of Toronto in 1929. He interned at the Grasslands Hospital in New York from 1929 to 1930 and served a residency at the Bellevue Hospital in New York from 1930 to 1932.

Dr. Logan settled in Lovell, Maine and practiced medicine there until 1934, then moved to the Portland area where he became a member of the X-ray Department of the Mercy Hospital, later serving as chief of that department. He did postgraduate study at the University of Pennsylvania from 1936 to 1940 under Eugene Pendergrass, M.D., receiving a Board qualification in Radiology. He returned to Portland and served as a member of the Executive Committee at the Mercy Hospital for over 20 years and became president of the Medical Staff in 1953.

Dr. Logan was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, the Radiological Society of North America, the New England Roentgen Ray Society, was a diplomate of the American Board of Radiology and served as president of the Maine Radiological Society in 1955 and counselor in the American College of Radiology in 1959. He also served as consultant at the Bath Memorial Hospital, the Brunswick Community Hospital and the Webber, Notre Dame and Trull Hospitals in Biddeford.

Surviving are his widow, Vivian D. Logan, two sons and a daughter.

Jack Manol, M.D.
1922-1965

Jack Manol, M.D., 42, of Portland, Maine died on August 17, 1965. He was born on the island of Crete on December 20, 1922, son of Emanuel and Athena Manolikakis.

Dr. Manol graduated from the Lyceum in Athens, Greece in 1940 and received his medical degree from the University of Athens in 1948. He interned in Athens from 1950 to 1951 and served a residency in Athens from 1951 to 1954 and in New York from 1954 to 1957. He attended Columbia University after coming to New York City in 1953. In New York he was associated with Sea View Hospital, Staten Island; Poly Clinic in St. Luke's Hospital, Manhattan and the Hospital for Crippled Children, Brooklyn, New York. He moved to Portland in 1958 and practiced medicine there until his death.

He was a member of the Cumberland County Medical Association, the Maine Medical Association and the American Medical Association. He was also a member of the Crippled Children's Clinic of Maine, the Portland Medical Club, the American Association of Physicians and Surgeons, the American College of Surgeons, the Orthopedic Society of Greece and the American Red Cross in Athens, Greece.

Surviving are his mother; his widow, Mrs. Irene Manol; a daughter, Miss Joanne A. Manol; four brothers, Ristedes, John, Minas and Emanuel Manol, Jr., all of Greece and a sister, Miss Mary Manol of Crete.

Howard R. Ives, M.D.
1911-1965

Howard R. Ives, M.D., 54, of Portland, Maine died on August 25, 1965. He was born in Cape Elizabeth, Maine on May 14, 1911, son of the Reverend Hilda L. Ives and the late Howard R. Ives.

Dr. Ives graduated from Deerfield Academy in 1929, Williams College in 1933 and received his medical degree from Yale University School of Medicine in 1937. He interned at the Rhode Island General Hospital from 1938 to 1940, was a fellow in surgery at the Mayo Foundation in Rochester, Minnesota from 1940 to 1941 and was on the surgical staff of St. Bartholomew's Hospital in London, England from 1941 to 1943.

Dr. Ives was the first American doctor to be accepted for service in Great Britain during World War II, where he served under Sir James Patterson, surgeon to the Queen. He also served for thirty-three months in the U. S. Navy Medical Corps in the Pacific. Dr. Ives located in Portland in 1946 where he practiced surgery until his death.

He was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, the American College of Surgeons and the New England Surgical Association. Dr. Ives was also a diplomate of the American Board of Surgery and was senior surgical attendant on the staffs of the Maine Medical Center and Mercy Hospital.

Surviving are his widow, the former Sally Means; his mother; a son, Howard Rollin Ives, III; two daughters, Sally and Sydney Ives and two sisters, Mrs. John E. Palmer, Cumberland and Mrs. Lawrence Boyer, Los Angeles, California.

Daniel M. Rowe, M.D.
1893-1965

Daniel M. Rowe, M.D., 72, of Scarborough Beach, Maine died on September 2, 1965. He was born in Portland, Maine on June 14, 1893, son of William J. and Catherine E. Rowe.

Dr. Rowe graduated from Portland High School, attended the University of Maine, Wesleyan University, Dartmouth College, and received his medical degree from Jefferson Medical College in 1926.

Dr. Rowe served in the Aviation Section of the U. S. Signal Corps during World War I and returned to Portland following the war where he practiced medicine until his retirement in 1959.

Dr. Rowe was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, the Mercy Hospital staff, the local board of Veterans Administration, the American College of Physicians and Surgeons and Harold T. Andrews Post American Legion. He was also a former member of the Knights of Columbus and the Portland Lodge of Elks.

Surviving are his widow, the former Bridie O'Connor; four sons, Daniel, Franklin, Massachusetts; Dennis, Long Beach, California; David, Mystic, Connecticut and Midshipman Donald Rowe of the U. S. Naval Academy; a brother, Joseph T. Rowe of Mexico City, Mexico; a sister, Mrs. George Foley of Portland; seven grandsons and several nieces and nephews.

Charles M. Armstrong, M.D.
1883-1965

Charles M. Armstrong, M.D., 82, of Robbinston, Maine died on September 11, 1965. He was born in Calais, Maine on July 4, 1883, son of Edward and Augusta Armstrong.

Dr. Armstrong attended schools in Calais and was first employed in his uncle's drugstore at St. Stephen, N. B. He later became a registered pharmacist and worked his way through Tufts University School of Medicine where he received his medical degree in 1915.

Dr. Armstrong practiced medicine at Woodland before mov-

ing to Robbinston where he had practiced for the past 49 years.

Dr. Armstrong was an Honorary member of the Washington County Medical Society and the Maine Medical Association, having received a 50-year pin in June 1965. He was also a member of the American Medical Association.

Surviving are his son, Charles M. Armstrong, Jr. of Bangor; two nephews, six grandnieces and three grandnephews.

County Society Notes

OXFORD

A business meeting of the Oxford County Medical Society was held on June 9, 1965 at Bethel Inn in Bethel, Maine which was followed by a social hour and dinner.

Harold L. Osher, M.D., of Portland, the guest speaker, gave a talk on clinical diagnosis of cardiac disease. Dr. Osher's excellent presentation was illustrated by lantern slides, x-ray films and film strips.

AKE AKERBERG, M.D.
Secretary

SOMERSET

The annual meeting of the Somerset County Medical Society was held at the Colony House in Lakewood, Maine on August 17, 1965. The Vice-President, H. Carl Amrein, M.D., called the meeting to order.

Clement A. Hiebert, M.D. of Portland, the guest speaker, was introduced by the program chairman, Richard P. Laney, M.D. Dr. Hiebert presented a most interesting program telling of his trip to West Africa earlier this year, during which he joined other personnel on the ship HOPE anchored in the harbor of Conakry, Guinea. Colored slides were presented and of particular interest were the slides showing the famed Dr. Schweitzer and his hospital "colony" in Lambarene.

John F. Dougherty, M.D., President of the Maine Medical Association, spoke briefly relative to the Maine Medical Education Foundation, a financial aid program established in 1960 to assist in the medical education of worthy students. Dr. Dougherty asked the members for continued assistance to this program. Following these remarks there was a brief discussion of the medical needs of Maine; especially the need for general practitioners.

The Nominating Committee consisting of Drs. George E. Sullivan, Edgar J. Smith and John P. Dow presented a slate of officers for 1965-1966 which was approved.

President, Marian L. Strickland, M.D., Canaan
Vice-President, Carlton E. Swett, M.D., Skowhegan
Secretary-Treasurer, John P. Dow, M.D., Pittsfield
Delegate to the Maine Medical Association House of Delegates: Richard P. Laney, M.D., Skowhegan. Alternate: Harland G. Turner, M.D., Norridgewock
Board of Councilors, Drs. John P. Dow, Howard L. Reed, Skowhegan and Franklin P. Ball, Bingham
Program Committee, Drs. Richard P. Laney, Edgar J. Smith, Fairfield and H. Carl Amrein, Madison
Discipline and Ethics Committee, Drs. Franklin P. Ball, William B. Grow, Fairfield and Henry E. Marston, North Anson

MARIAN L. STRICKLAND, M.D.
Secretary

Deceased

PENOBSCOT

Arthur N. Lieberman, M.D., 180 Broadway, Bangor, Maine, September 27, 1965.

News, Notes and Announcements

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine
July 13-15, 1965

THROUGH EXAMINATION

Marcel Carbonneau, M.D., 3070, Lechasseur, Ste-Foy, P.Q., Can.
Carl Cornil, M.D., 105 Longwood Ave., Brookline, Mass.
William H. Crosby, Jr., M.D., Walter Reed Army Medical Center, Washington, D. C.
Ruben Fialkoff, M.D., 39 Haynes St., Manchester, Conn.
Efrain C. Galarraga, M.D., Augusta State Hospital, Augusta, Me.
Barbara Geldner, M.D., 6 Lenox St., Edison, N. J.
Nasser Ghassemi, M.D., 430 East 67th St., New York, N. Y.
Juta Helm, M.D., 376 Springville Dr., Buffalo, N. Y.
Miguel Hermoso, M.D., Augusta State Hospital, Augusta, Me.
Erwin F. Hirsch, M.D., Washington Hospital Center, Washington, D. C.
Kei H. Howes, M.D., 7 Grenville Rd., Watertown, Mass.
Douglass H. Howry, M.D., 166 Washington St., Marblehead, Mass.
Adnan T. Kaissi, M.D., 393 State St., Bangor, Me.
Mehrdad B. Khan-Malek, M.D., 353 Park Ave., Paterson, N. J.
Takanori Kimura, M.D., 630 West Washington St., Suffolk, Va.
John Madaras, M.D., Bangor State Hospital, Bangor, Me.
Fawzy S. Mansour, M.D., 1078 Lanier Blvd., Atlanta, Ga.
Majid A. Qureshi, M.D., St. Mary's Hospital, Cincinnati, Ohio
Dorothy A. Wescott, M.D., 1 Emerson Place, Boston, Mass.

THROUGH RECIPROCITY

William L. Budd, M.D., 30 MacMillan Dr., Brunswick, Me.
David M. Bull, M.D., North Haven, Me.
Willard H. Clark, M.D., 632 Main Ave., Twin Falls, Idaho
William W. Conner, M.D., Boothbay Harbor, Me.
Howard R. Deming, M.D., Portland, Me.

Paul J. Driscoll, M.D., 103 New Jersey Ave., Fort Dix, N. J.
John F. Egan, M.D., U.S. Naval Hospital, St. Albans, N. Y.
Walter B. Goldfarb, M.D., 370 Baxter Blvd., Portland, Me.
Donald M. Good, M.D., 49 Marks St., St. Stephen, N. B., Can.
George W. Hardt, M.D., 295 Delavan Ave., Bryam, Conn.
Richard C. Hirschhorn, M.D., Hancock Point, Me.
Thomas E. Lally, M.D., Augusta State Hospital, Augusta, Me.
Joseph M. Laltoo, M.D., York Hospital, York, Me.
Chris A. Lutes, M.D., RFD #2, Stephenson St., Cape Elizabeth, Me.
Warren R. Nickerson, M.D., 137 Fairview, Elmhurst, Ill.
David L. Phillips, M.D., Box 418, Rumford, Me.
Robert F. Ritchie, M.D., Maine Medical Center, Portland, Me.
Harold W. Schell, Jr., M.D., Uncas on Thames Hospital, Norwich, Conn.
Ronald A. Swanson, M.D., 26 MacMillan Dr., Brunswick, Me.
Michael J. Taylor, M.D., P. O. Box 42, Westbrook, Me.
Robert M. True, M.D., 506 Boston Post Rd., Weston, Mass.
Channing H. Washburn, M.D., 640 Pleasant St., Paxton, Mass.
William A. Yorzyk, M.D., 1974 Allen St., Springfield, Mass.
Paul C. Young, Jr., M.D., Wentworth Douglass Hospital, Dover, N. H.

Meeting in Miniature on Steroids Presented by The Central Massachusetts Section of The American Chemical Society

Date: October 29, 1965 *Time:* 9 A.M. - 10 P.M.
Place: Alden Hall, Worcester Polytechnic Institute, Worcester, Massachusetts
Registration Fee: $3.00 (*Student Fee:* $1.00)
Luncheon $3.00 Dinner $4.00

IX International Cancer Congress October 23-29, 1966 — Tokyo, Japan

In connection with the IX International Cancer Congress, which will take place in Tokyo, Japan, October 23-29, 1966, a charter flight and several group flights are contemplated, thus substantially reducing the cost of transportation.

Members of the immediate family of congress participants will be eligible.

Anyone interested should communicate with Hirsch Marks, M.D., 435 East 57th Street, New York 22, N. Y.

Book Reviews

Control of Glycogen Metabolism (Proceedings of an International Symposium held July 23-25, 1963). Edited by W. J. Whelan, Ph.D.; D.Sc.; F.R.I.C. and Margaret P. Cameron, M.A. London, England, 1964. Little, Brown and Company, 434 pp, $12.50.

This symposium on glycogen metabolism is highly technical, dealing with the structure of glycogen, glycogen derived oligosaccharides, branching enzymes, the structure of phosphorylase, the mechanism of actin and role of amylo-1,6 glucosidase, oligo-1,4→1,4 -glucantransferase, regulation of glycogenolysis in muscle, role of insulin and control of UDPG alpha glucan transglucosylase activity; possible indirect effects of adrenalin on glycogen metabolism; a discussion of hexokinase and glucokinase.

The section on glycogen storage disease is most useful to the clinician and probably the only section of the symposium in which he will feel at home. Glycogen storage diseases are grouped into 6 types; Von Gierke's disease, type I; Pompe's disease, type II; Cori's, Forbe's disease; limit dextrinosis, type III; Andersen's disease, type IV; McArdle's disease, type V; Hers's disease, type VI.

Clinical manifestations range from hepatomegaly, cardiomegaly, hypoglycemia to muscle cramps. Of great clinical interest is a description of glycogen synthase deficiency (UDPG - glycogen -1,4- glucosyl transferase) occurring in one family manifested by profound fasting hypoglycemia. Liver glycogen stores are very low, and glucose tolerance is reduced. The adrenal medulla does not respond to hypoglycemia.

The final discussion deals with the collection, size, and storage of tissue samples. This is extremely important and often neglected. Emphasis is placed on rapid freezing by dropping the sample directly on dry ice. Other more complex freezing methods are described.

The chapters in the symposium are almost uniformly well written and concise; some are exceptionally lucid. There are many useful tables and graphs. This is an outstanding, authoritative textbook that will have the widest appeal to research biochemists and physiologists. The clinician will find it a very valuable reference text, if he has problems of disturbed glycogen metabolism.

PETER F. LANSING, M.D.
Augusta, Maine

Surgery of the Biliary Passages and Pancreas. By Dr. Walter Hess, Privatdozent in the Surgical Department of the University of Basel, Switzerland. Published by D. Van Nostrand Company, Inc., Princeton, New Jersey. 638 pages with illustrations. Price $25.00.

This book represents a thorough evaluation of one man's extensive experience with biliary tract and pancreatic problems and also very clearly expresses the author's opinions in many situations where total agreement on the subject is far from complete. An excellent review of physiology and anatomy of the region is had as well as the diagnostic aspects of biliary tract disease. The author is also considerably interested in the field of x-ray interpretation of the biliary tree and also visual observation of the bile ducts through an endoscope and his approach to this problem certainly warrants further investigation as he has presented some very fascinating x-ray and film pictures taken during cholangiographic studies of the lower biliary tract. His interpretation of the functions of the sphincter of Oddi are extremely interesting. Whether or not his particular opinion as to the interpretation of these films will hold up, only time will tell. At least he has taken a very positive step in this direction.

The surgical orientation of the book is well founded. Many American authors will have some reserve about his interpretation of the need for sphincterotomy. He certainly has utilized this widely for numerous conditions, but in general the book represents a well planned approach by an individual author to present his rather extensive experience in the field. The book would certainly be of value to people specifically interested in this area of surgery and also as a reference text for medical libraries. The most divergent aspect of this book is use of x-ray studies during the course of biliary tract surgery, not only to delineate stones and stricture but to try to correlate pressure studies and photographic studies of the common duct and papilla in action and then act accordingly with his interpretations of the findings. Readers of this book will gain a great deal of knowledge of surgery of the biliary passages and pancreas.

JAMES L. FIFE, M.D.
Brunswick, Maine

The Journal
of the
Maine Medical Association

Volume Fifty-Six | Brunswick, Maine, November, 1965 | No. 11

Anesthesiologist and Hemorrhagic Shock

C. Paul Boyan, M.D.

The pathologic physiology of the various types of shock has been studied extensively over the past 20 years. Unfortunately the factors involved in producing shock in experimental animals and those observed at the bedside did not appear consistently to be identical, or more often the treatment did not seem to have the same or similar results. Hemorrhagic, or better oligemic shock, as a clinical entity, is of particular interest to the anesthesiologist.

The main feature of oligemic shock is decreased tissue perfusion because of ineffective microcirculation. See Fig. 1. Loss of whole blood or plasma produces the following cardiovascular derangements: vasoconstriction; reduction of cardiac output, blood pressure and coronary blood flow, with subsequent myocardial depression; inadequate tissue perfusion with cell hypoxia leading to acid-base balance changes toward metabolic acidosis. This acidosis further depresses the cardiac function thus establishing a dangerous vicious circle. Studies have shown that the blood flow in the microcirculation is compromised during shock; there is red cell aggregation (sludging) and an increase in blood viscosity.[1] All these physiologic alterations, unless corrected, will progress until an irreversible state is reached and death is inevitable. The clinical signs of oligemic shock are well known and do not have to be repeated. In brief, the bleeding patient is vasoconstricted, hypoxic, acidotic and with compromised microcirculation.

The anesthesiologist who is called upon to treat or anesthetize a patient who is in oligemic shock or may suffer severe blood loss, faces a challenging problem. It should be noted that no single test or measurement will give the answer to what should be done and how. Absolute values of many tests can be misleading; they should be done serially and above all correlated with the clinical picture. Our endeavors should be not to treat separate signs and symptoms but the patient as a whole. This cannot be stressed strongly enough.

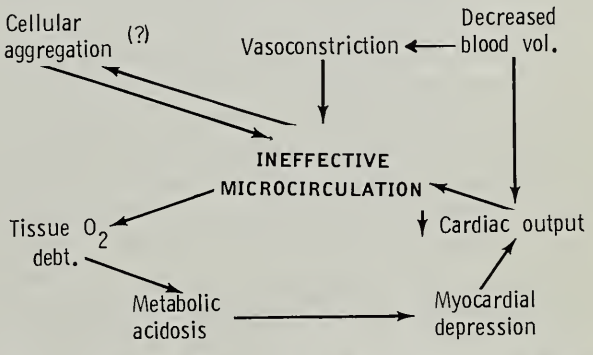


Fig. 1. Relationship of factors producing ineffective microcirculation in hemorrhagic shock.

Obviously the most urgent task is to stop the continuing hemorrhage by surgical intervention which can vary in scope from a simple ligation of a vessel to extensive surgery. It has been our experience that if the bleeding has not been controlled the patient will die in spite of all heroic measures.

The establishment of an adequate airway, pulmonary ventilation and oxygenation plays an important part in the treatment of the bleeding patient. This may require nothing more elaborate than proper positioning of the head and mandible or clearing the pharynx from secretions, vomitus or blood and administration of oxygen through a nasal catheter or mask. At times, endotracheal intubation, or tracheostomy may be necessary to secure an unobstructed airway. Adequate pulmonary ventilation often needs manual or mechanical assistance and may depend upon relief of hemothorax, pneumothorax

From the Department of Anesthesiology, Memorial Sloan-Kettering Cancer Center, New York, New York

or stabilization of flail chest. Collection of blood in the abdominal cavity will in due time splint the diaphragm to such an extent that normal expansion of the lungs will be impossible.

To restore normal blood volume and correct the circulatory derangements is the logical approach to treatment of oligemic shock. The following points are of clinical importance:

It is very difficult to measure the exact blood loss. A satisfactory approximation can be derived by weighing the blood soaked sponges and lap pads and measuring the contents of suction bottles providing it is done expeditiously by a designated person.

Citrated blood, although the best available substitute for lost blood, is not the most physiological replacement. Bank blood has the following characteristics: low pH (around 6.6) from a combination of metabolic and respiratory acidosis; the pCO_2 is increased to 180 mm of Hg; the acid load is about 7 mEq due to accumulation of lactate, pyruvate and citrate; hematocrit averages 41%; low temperature (4-6°C.); increased concentration of free hemoglobin, K, Na; deficiency in Ca^{++}, platelets and clotting factors V and VIII. See Table 1.

If the bank blood is given cold and in large amounts to a patient who is already in shock, the existing vasoconstriction and acidosis of anaerobic metabolism will be maintained by hypothermia and the exogenous acid load of blood transfusion. Thus the patient will remain in shock in spite of the efforts to restore normal blood volume. Therefore, the cold bank blood should be warmed to body temperature while being transfused. This is easily done with a simple blood warming device. See Fig. 2. In this way the replenished blood volume will improve the cardiac output, and restore the vital microcirculation. The tissues will be able to metabolize rapidly the citric acid supplied by the bank blood, the endogenous lactic and pyruvic acids and correct the existing metabolic acidosis. Patients receiving blood through the warmer exhibit the signs of normal circulation; they are warm, dry, pink and with easily obtainable blood pressure.[2] In emergencies plasma or dextran can be given until bank blood is made available.

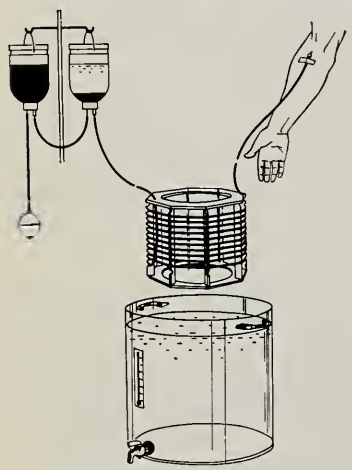


Fig. 2. Blood warmer incorporated into transfusion setup during massive blood replacement.

It is our belief that patients with normal calcium metabolism, except infants, do not need the addition of Ca.++[3] The body has a huge reservoir of calcium in the bones and can draw from it when the need arises. The combination of hypothermic heart and exogenous

UNIT OF BANK BLOOD

120 ml ACD solution. 480 ml blood
Hct. 41%. Stored at 4°C
pH-6.6. pCO_2 180 mm Hg
ST. Bicarbonate 5 mEq/L
Acid load 7 mEq
No plasma Ca^{++}, platelets
↓ Plasma factors VIII V (labile)
↑ Plasma HB and K (7 - 23 mEq/L)

Table 1. Characteristics of ACD preserved bank blood.

Ca^{++} can produce cardiac arrhythmias and cardiac arrest with ventricular fibrillation. We have not seen this complication since we stopped six years ago administering Ca gluconate during blood transfusions.

The inherent acid load of bank blood which is about 7 mEq. per unit should be buffered by simultaneous administration of sodium bicarbonate. We use 44.4 mEq. per 3000 ml. of bank blood. If the hemorrhage has occurred in the operating room and blood transfusion has been started right away, bicarbonate should be administered only when the signs of shock are evident since at this point the body buffering system, trying to combat the metabolic acidosis of poor tissue perfusion, should not be taxed with additional exogenous acid load. If normal microcirculation has been maintained with transfusion of warmed blood, there is no need for buffering unless large amounts, 15 or more units of bank blood, have been given rapidly.

Serial microhematocrit determinations will help to approximate the circulating red blood cell mass. We and other investigators have observed a drop in hematocrit with rapid loss of at least 10-15% of the blood volume. The body, especially under anesthesia, tries to compensate for the lost blood by moving fluid from the extracellular space into the vascular compartment. At times, patients could lose predominantly plasma and have low blood volume with a high hematocrit while demonstrating signs of shock. In the first instance the therapy will be transfusion of whole blood or even packed cells while plasma administration is indicated when the hematocrit is high. Serial microhematocrits, in addition to the clinical status of the patient, will help to follow the progress of the treatment. This test is fast, inexpensive, easy to do and has proven of value in our experience. Recent surgical literature has repeatedly emphasized the inadequacy of the hematocrit and the importance of blood volume in the assessment of the need

for blood replacement. It is generally stated, however, that changes in hematocrit resulting from the dilution occur very slowly. Actually they are rapid as found by Doberneck and others.[4]

Blood volume studies became practical with the use of a semi-automatic computer and tagged albumin tracer RISA. A valid objection to the accuracy of blood volume determinations in shock is raised because of poor mixing of the tracer during impaired blood circulation leading mostly to higher values. However, a low figure as compared to standards or relative to previous determinations will indicate the need for more replacement. A high figure, on the other hand, should not stop replacement therapy if the signs of deranged circulation persist. We like to use blood volume determinations as a check of our clinical judgment in blood replacement. McLean[5] has found that in the treatment of shock the effective blood volume or that volume which restores cardiac output, blood pressure, urine flow and tissue perfusion always exceeds normal blood volume and for this reason blood volume determinations may be misleading rather than helpful.

The blood sample for hematocrit and blood volume studies should be taken from a big vein or artery. A disposable 21 gauge needle attached to a vacutainer will do no lasting damage to the wall of a larger artery. Blood samples taken in this way are suitable for acid-base studies as well. The pH, pCO_2 and standard bicarbonate can be measured rapidly by the Astrup method. Knowing the base line of the acid-base balance will help in assessing the status of the patient and the progress of treatment.

Central or peripheral venous pressure monitoring is a good guide of the patient's circulating blood volume and cardiovascular status providing it correlates with the overall clinical picture. A proper interpretation of venous pressure requires experience since it could be influenced easily by various factors. For instance: interference with the venous return such as pressure on vena cava by packs or retractors will lower the central venous pressure although the blood volume is normal; change in patient's position without adjusting the zero point or partial occlusion of the catheter or needle openings will give false readings. Low blood volume may go along with high or even normal central venous pressure if the heart is beginning to fail.

The use of vasodilators to improve the tissue perfusion as advocated by some investigators, mainly Nickerson[6] is a very delicate intervention. In our opinion, low blood pressure due to vasodilation does not mean better flow and tissue perfusion. It is easy to imagine what will happen to a patient who has a low blood volume and in whom the vascular tree has been suddenly expanded. We have done acid-base studies in patients subjected to deliberate hypotension with short acting ganglionic blockers and have found an increase in metabolic acidosis and excess lactate. Therefore, this technique is still under investigation and requires experience not easily acquired.

	Diethyl ether	Halothane	Methoxyflurane	Cyclopropane	
Patients	51	34	31	39	No blood transfusion
Standard bicarbonate mean net changes mEq/L	-1.586 ± 1.51	-1.647 ± 2.054	-1.006 ± 1.751	-0.935 ± 1.43	
	-2.294 ± 1.351	-1.46 ± 1.459	-1.878 ± 2.139	-0.794 ± 1.431	Blood transfusion <2400 ml
Patients	39	20	19	18	

Table 2. Mean net standard bicarbonate changes during surgery and diethyl ether, halothane, methoxyflurane or cyclopropane anesthesia in patients without and with blood transfusion.

The prolonged use of vasopressors, or as we call them "embalming drugs," is outdated in view of the present concept of ineffective microcirculation as the main physiologic derangement in oligemic shock.

It has been stated that low molecular weight dextran improves the viscosity of human blood at low shear rates.[8] In addition, low molecular dextran has a favorable effect on the surface electrical charge of the erythrocyte, maintaining its negativity and mutual repelency thus combating aggregation. We have measured the blood viscosity in operated patients who have received low or high molecular dextrans and compared it with the base line. The dextrans brought about a short lived hemodilution with an insignificant change in viscosity.

A final word about the anesthetic agent in hemorrhagic shock. We have followed the acid-base balance in patients subjected to anesthesia and surgery of several hours duration and blood transfusion of up to 2500 ml. of bank blood. The following agents were studied: diethyl ether, halothane, methoxyflurane and cyclopropane. All four anesthetics produced metabolic acidosis of one to two mEq/L. as measured by the net changes in the standard bicarbonate of whole blood. See Table 2. These figures are within the daily human variations and are not of clinical significance providing the patient's base line was within normal limits. If the patient is in oligemic shock and acidotic, the anesthetic of choice will be the one which produces the least additional metabolic acidosis. Our figures show that there is no significant difference among the agents studied except when up to 2400 ml. of bank blood have been given, cyclopropane produces less metabolic acidosis than diethyl ether ($p<0.01$). Therefore, it seems prudent to use cyclopropane in patients who are in oligemic shock.

Sometimes after multiple blood transfusions patients demonstrate the so-called oozing tendency. All cut tissues bleed from everywhere and the blood has a watery consistency. Extensive studies have not found the main cause for this bleeding tendency.[9] Apparently subcritical deficiencies in several clotting factors are responsible for this syndrome. It is known that bank

blood is deficient in Factors V, VIII and platelets. Therefore, when oozing is diagnosed, fresh blood, fresh frozen plasma or in rare instances platelet concentrate should be administered. Presence of fibrinolysis will require the administration of epsilon amino caproic acid. It should not be forgotten that fibrinolysis is a temporary phenomenon and may be present without signs of a bleeding tendency and vise versa.[10] A simple observation of the blood clot formation, retraction and lysis in a water bath of 37°C. will give a rough idea of the clotting mechanism status. Clot lysis of less than 30 minutes is a sign of fibrinolysis and epsilon amino caproic acid should be administered.

Conclusion

Oligemic shock is a critical situation requiring intervention without delay. While the bleeding is controlled by surgery, good ventilation, oxygenation and rapid restoration of normal blood volume are the necessary steps for successful treatment. Bank blood should be warmed to body temperature and buffered with sodium bicarbonate. Microhematocrits, blood volumes, venous pressure, acid-base balance determinations are helpful tests which should be interpreted in correlation with the overall clinical picture. A failure to benefit from transfusion of blood after hemorrhage is often the result of undertransfusion.

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Services To Armed Forces Rejectees

On September 13, 1965 at the Bangor Armed Forces Examining Station, the Maine Department of Health and Welfare inaugurated its new Services to Armed Forces Rejectees Program. The program of counseling and referral of armed forces medical rejectees will also become operational at the Portland Examining Station by the end of November.

Governor John H. Reed designated the Department of Health and Welfare to supervise the Federally-financed project here in Maine; similar programs are operational in 39 of the other 50 states.

The service is one of counseling and motivation for treatment—not one of treatment. S A F R counselors in Bangor and Portland will talk to each medical rejectee at the time of his rejection. The counselor will stress the importance of having one's health needs met and will make the rejectee aware of the availabilities of assistance through treatment by private physicians, as well as official or voluntary agencies. If the rejectee is not currently under care and treatment, he can accept or decline the services of the counselor. For those rejectees who are under care and treatment, only a verification of such care will be made.

The counselor will make an appointment to see the rejectee within two weeks after the interview at the Examining Station if the rejectee desires possible referral. The program is based on the premise that referral to the private physician is the best course. Referrals to official and voluntary agencies will be made only after consulting with the attending physician whenever possible.

After referral, the rejectee will get a one-month follow-up and a six month detailed follow-up by the rejectee service counselor.

The program is independent of the Selective Service in that there is no requirement or intention to report the correction of defects to the Selective Service.

Edwin H. Pert is State Program Director with headquarters at 280 Front Street, Bath, phone 443-6570. The Portland counselor will work out of the Bath office.

A Bangor field office, staffed by counselor Paul Parks, is located at 205 French Street, Bangor, phone 947-7844.

Bleeding Renal Hamartoma

An Uncommonly Encountered Complication in the Tuberous Sclerosis Complex

J. Galindo, M.D.* and R. E. McAfee, M.D.**

The description of sclerotic cerebral areas occurring in young adults suffering from mental deficiency was first made by Bourneville in a series of papers published from 1880 to 1900; although prior to these, Von Recklinghausen, as early as 1862, had described cerebral tubers and renal tumors in some of his own cases.

Bourneville also included in his description the presence of renal hamartomas and sebaceous adenomas of the face, although he thought that the latter represented separate entities and that the association was fortuitous.

It wasn't until 1908 that Voght postulated that mental deficiency, epilepsy, cerebral tubers and cardiac and renal tumors were all integral parts of the same pathologic complex. (Tuberous Sclerosis).

The familial occurrence of Bourneville's disease was subsequently described by Berg in his study of a family of tuberous sclerosis patients over a period of 3 generations.

E. B. Sherlock, in 1911, coined the term of "epiloia" for the tuberous sclerosis complex which, curiously enough, was picked because it was completely meaningless.

A number of other authors have also described the location of tumors at other anatomical sites, such as the retina where the latter have been labelled as "phakomas".

Abnormalities in other organs have been encountered, such as the presence of dermal tags, pigmented and non-pigmented nevi, vitiligo, fibromas in ungual and subungual locations, cafe au lait spots, etc.

X-ray studies may demonstrate the presence of intracranial calcifications and of bony lesions of the skull, feet, hands and ribs; Hasegawa,[17] Beck and Hammond,[3] Ashby and Ramage,[2] Yakovlev.[40] In addition, a number of patients have shown developmental anomalies such as spina bifida and cleft palate.

Case Report

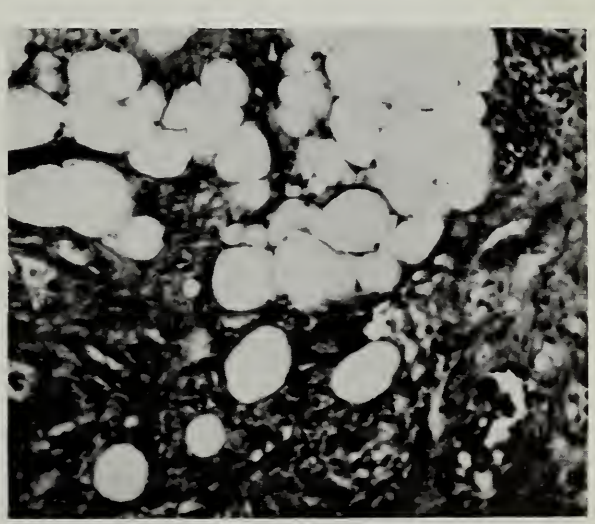
This 15-year-old white male with known tuberous sclerosis and mental retardation began vomiting the night prior to admission to the surgical ward. He complained of initial epigastric pain, progressing within a few hours to generalized abdominal tenderness. There were no associated urinary symptoms. A CBC done at the referring hospital revealed a normal hemoglobin and white count of 12,600 with 64% polys and 20% band forms with 12 lymphocytes and 4 monocytes. Initial impression was probable appendicitis.

On admission to the surgical ward his blood pressure was 120/80, pulse of 100, respiration 20, temperature 100.4° rectally. He was a well-developed, muscular white male with moderate abdominal pain and with obvious adenoma sebaceum of the face. Abdominal examination revealed: generalized tenderness with both subjective and objective localization in the right lower quadrant and right lower flank. There was no palpable mass. Bowel sounds were diminished in intensity. Rectal examination revealed mostly mild tenderness in the cul de sac without localization. Initial impression again was: probable appendicitis? urinary tract infection.

The plain film of the abdomen was taken and revealed the right renal shadow to be somewhat obscured by a questionable



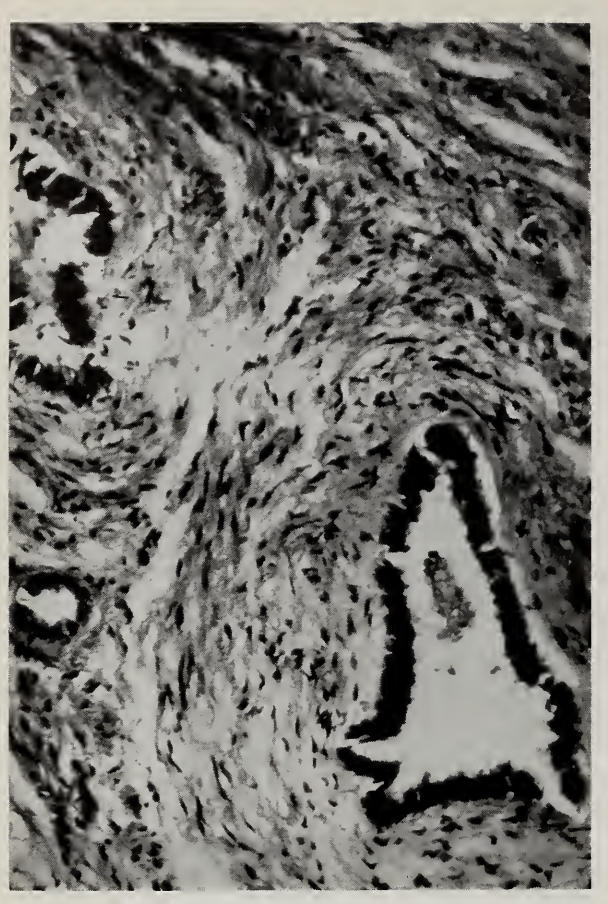
The two types of cells described in the text are well-evidenced here, the small dark cells with rounded nuclei and small amount of cytoplasm and the larger cells with vesicular nuclei and ill-delineated nuclear membranes. X 120.



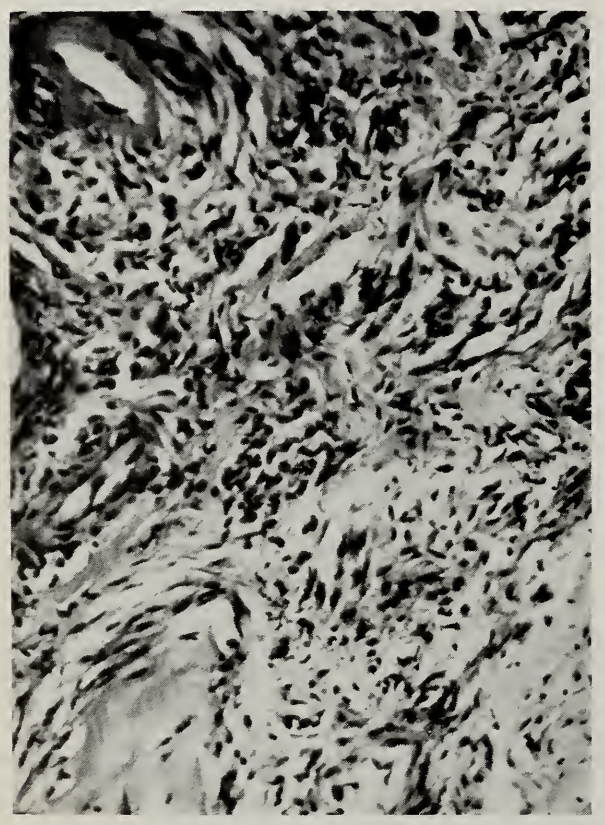
Lipoid cells, undifferentiated small and large cells, vessels, and one tubular element on lower right. X 120.

*Director of Pathology, Pineland Hospital and Training Center, Pownal, Maine

**Chief Surgical Resident, Maine Medical Center, Portland, Maine



Fairly well differentiated tubules surrounded by connective tissue elements. X 120.



Numerous vessels with patent lumina are separated by small dark fibrocytes which in areas form curlicues. X 120.

mass in the area. Because of progressive and increasing pain predominantly in the right lower quadrant along with a continued elevation of the white blood count, a laparotomy was carried out. On opening the peritoneal cavity some 500 to 700ml. of free blood were present. The appendix was normal. Exploration revealed the presence of an actively bleeding mass in the right renal fossa which had perforated through the posterior peritoneum afterwards leaking into the peritoneal cavity. Further dissection of this area revealed the kidney to be replaced by a large lemon-sized mass arising from the lower pole of a small atrophic kidney and hemorrhage was seen issuing from this mass. A right nephrectomy was carried out removing the tumor in toto plus the right ureter. Palpation of the left kidney revealed a small nodule on the upper pole, but was otherwise felt to be normal. The patient had an uneventful recovery. A postoperative IVP was performed which showed the remaining left kidney to be somewhat larger than normal. There was prompt excretion and good concentration of dye. No obstruction of the ureter and bladder were evident and the conclusion was that of a normally functioning left kidney and ureter with normal bladder and surgical absence of the right kidney.

Histopathology

The renal tumor consisted of a moderately firm, partially encapsulated mass about 4.5cm. in greatest diameter which presented some yellow, fatty, ill-delineated areas and a few varying size zones of peripheral hemorrhages.

Haphazardly scattered, there were well formed renal tubules within a connective tissue stroma which was well vascularized. Aggregates of small round nucleated cells with a small rim of cytoplasm were evident as were somewhat larger cells with vesicular neuclei and a wide cytoplasmic rim with an indistinct cellular membrane.

Towards the periphery of the tumor there were a few hemorrhages. The areas bordering on them bore evidence of expansive compression evidenced by flattening and elongation of the cellular constituents. Both the small and the large cells described above formed irregular varying size curlicues. Here and there aggregates of lipoid cells were seen.

Summary

A case of a 15-year-old white male with known tuberous sclerosis and mental retardation presented with signs and symptoms of acute appendicitis. At surgery a bleeding hamartoma of the right kidney was removed. The case is presented as a previously recognized but seldomly encountered problem associated with the tuberous sclerosis complex.

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Hypnosis In General Practice

WARD A. ALBRO, M.D.

One of the most often heard excuses for not using hypnosis in general practice is that it must be time consuming. Nothing could be farther from the truth. In many cases one or two short sessions are sufficient to produce the desired results. The actual time involved may run from 15 minutes to one hour. This makes it a practical and useful modality which is very applicable to general practice as well as to the specialties.

It is in no way suggested that hypnosis be used in general practice alone, but it has a definite place along with a complete history, physical examination, appropriate laboratory and x-rays, chemotherapy, and psychotherapy. While the main theme of this paper is to show that hypnotherapy can be successfully used in general practice without excessively long office visits, it will also become apparent from the following short case histories that it is an effective means of reducing and/or eliminating much of the use of tranquilizers and analgesics which may have noxious side effects as well as habit forming potentialities.

The techniques of trance induction used were for the most part a progressive relaxation similar to that described by Wolberg[1] and/or the "clenched fist" technique modified from Stein.[2] Direct symptom removal was utilized effectively without replacement symptoms developing which is in accord with the beliefs of Erickson,[3] a psychiatrist, who does not share the criticisms leveled by some psychiatrists that direct symptom removal is fraught with dangers.

CASE 1

This patient, age 26, gravida III, para III, was first seen at home with a severe left-sided headache associated with slight nausea, but no scotomata. She had been taking aspirin gr. X-XV every three hours without relief. Examination revealed only an irregular pulse with frequent extrasystoles and a systolic pulmonic murmur which had been present for years. The patient was given Darvon® Compound and was asked to make an appointment in the office in a few days for hypnotherapy. Four days later the patient came to the office in a highly agitated state and said that she "felt as though she was going to fly apart". Her head was aching again on the left side. A progressive relaxation induction of trance was used. The patient appeared to be in a very light trance and this was deepened by asking her to count backward from 100 very slowly and to breathe slowly and deeply, and that with each breath she could become more and more relaxed. On arousing herself she stated that she felt much more relaxed and that her headache had almost disappeared. Her total time in the office was 20 minutes. She was asked to return in about a week for more hypnotic relaxation and was seen again in 11 days. She entered a trance state easily on closing her eyes and breathing deeply. A glove anesthesia of the right hand was suggested and obtained, and she was told that this pleasant pain-free state of her hand could be transferred to her head. This was accomplished and relief obtained. A posthypnotic suggestion was given that she could employ this technic at home. Further history revealed that she had been "very nervous" all of her life and that this was aggravated whenever she was in large groups of people such as at PTA meetings, etc. This second session required 25 minutes. One month later a follow-up phone call showed that she had been feeling well and had no more headaches. She was employing self-relaxation regularly. She felt that there had been a marked improvement in her "nervousness" and that she was more relaxed in crowds. She further stated that if she had a return of her tenseness or headaches she would like to return for more hypnotherapy.

CASE 2

A married housewife, age 37, was seen with the request that hypnosis be used to help her lose weight. A complete physical examination was done which was within normal limits except for moderate obesity which was confined to the hips and abdomen. She requested that she lose weight but stated that she just wanted to lose it on her hips and abdomen, but did not want to decrease her bust size. Her pre-treatment hip measurement was 38 inches and she hoped to reach 36 inches. This patient was one of my first attempts with hypnosis and an eye-fixation (Wolberg[1]) technic was used. The patient was intelligent and cooperative, but she did not allow her eyelids to get heavy so she was asked to close them. Progressive relaxation was then suggested with moderate success. From time to time she opened her eyes and was each time asked to close them. At no time was she in much more than a very light hypnoidal state, but, in spite of this, suggestions were given to practice self-relaxation at home for 15 to 20 minutes a day and during this time to visualize how she would like to appear and how she would like to have her clothes fit. Office time required for hypnotherapy was about 20 minutes. She was seen one month later and was very pleased with her progress having lost one inch from her hips and her abdomen had become flatter. No formal induction was done at this visit. It was suggested that since she was doing so well she could continue self-relaxation and visualization and could anticipate reaching her desired weight and measurements in a month. Her office visit on this occasion was only 15 minutes.

About one month later she called me on the phone to say that her hips measured 36 inches, that her clothes fit well, and that her bust measurement had not changed. Six months later she was maintaining her weight with no difficulty.

CASE 3

This patient, age 22, married gravida I, para I, came in the office with multiple complaints. Her chief complaint was severe abdominal cramps and constipation. She also complained of being overweight, nervousness, and sterility. A physical examination and barium enema were done and no organic disease was found. She had been seen by several physicians over the past few years for the complaint of severe left-sided abdominal cramps and had had no relief of her symptoms.

She was then seen for 25 minutes of hypnotherapy. Progressive relaxation was used with good results. Catalepsy was suggested and carried out. A glove anesthesia was then produced and it was suggested that this pain-free state in her hand could be transferred to her abdomen relieving her abdominal cramps. Further suggestions were given concerning her diet, i.e., to eliminate sweets, potatoes, and foods made with flour. She was also asked to eat more slowly and consequently to enjoy her food more fully and so to become satisfied with a smaller intake which would automatically limit her calories. Aroused from her trance she said that her abdominal pain had disappeared.

One week later she was seen for a half hour. She had had only occasional abdominal cramps and felt much less nervous. Her weight had dropped two pounds. Trance was induced by asking her to make herself comfortable and to enter hypnosis at her own rate. While in light trance more history concerning her nervousness was elicited. It was learned that her parents had been separated and she had been brought up by her mother. She had always been bashful and retiring and was bothered by crowds. It was suggested that by practicing self-relaxation at home she could improve her ability to relax and would arouse feeling very calm and that this feeling could persist throughout the day. Also that as she learned to relax her external muscles the inner organ muscles could relax and function more normally. Such inner relaxation and improvement of organ function could relieve tubal spasm and increase her chances for conception.

Her next visit was about 3 weeks later and lasted 25 minutes. During this time she had had no abdominal cramps, but had had difficulty sleeping because of "bad dreams". Questioning in more detail showed that this was not a new symptom replacing the abdominal pain, but it had been present for some time but had not been mentioned due to the severity of the abdominal pain. She also complained that she had been staying on her diet but had gained a pound. She had had several crying spells and feelings of depression. Again she was asked to close her eyes and relax as completely as possible. Her weight program was reemphasized and she was asked to imagine herself at the weight she desired. She was asked to hallucinate a pleasant experience; a shopping trip was chosen. This she was able to do quite vividly. It was suggested that she could have pleasant dreams instead of the disturbing ones and that she could awaken in the morning refreshed rather than depressed.

A month later during a 15 minute session she stated that she had had no further disturbing dreams and had not felt like crying. The reason for the weight gain was answered by a positive AZ Test! She was very pleased with her progress in the few short sessions; she had stopped having severe cramps, was much less nervous and had become pregnant which she had been trying to accomplish for two years.

Case 4

A married oil burner repairman, age 25, came to the office with the chief complaint of severe right-sided headaches. The headaches were partly relieved by aspirin. He state that he had always been "very nervous". The headaches came on suddenly both at work and on weekends when "he was relaxed as he ever could be. Examination was negative and he was given a mild tranquilizer for ten days with no relief.

He was then seen for a 25 minute session of hypnotherapy. Progressive relaxation was used to induce hypnosis. A glove anesthesia was suggested and developed; the patient tested it to his own satisfaction. It was then suggested that he could let his subconscious transfer this pain-free feeling to the side of his head which had been aching when he came into the office. On arousal he stated that he felt good and refreshed and that his headache was gone. It was suggested he practice this at home. Four months later he called saying he had had no more headaches and felt much more relaxed most of the time.

Case 5

A married housewife came with the chief complaint of irregular menstrual periods. Routine examination was within normal limits except for an enlarged and tender left ovary. She also admitted to dyspareunia on the left side and complained of increased pain after the pelvic examination. The patient admitted to being always very "nervous" and was very apprehensive about any surgery.

An induction of trance was started with progressive relaxation, but she exhibited so much tenseness and agitation that she was asked to concentrate her tension in her "clenched fist", according to Stein's technic. This produced greater relaxation and it was suggested that she did not have to have any pain in the left ovary since it was going to be corrected at surgery. On arousal from trance she stated that her pelvic pain was gone and she felt nicely relaxed. Surgery could not be scheduled for a week. Her total time in the office exclusive of history and physical examination had been 15 minutes. She was not seen again until the night prior to surgery. At this 15 minute visit it was suggested, without any formal trance induction, that surgery would be uneventful, that she would have minimal discomfort in her incision, and that all her normal functions of bowel and urinary tract would be no problem to her. She was also instructed to close her eyes after her preoperative medication "in order for it to work more effectively".

On the morning of operation she came to the operating room very calm and relaxed. Her postoperative course was unusual in that she required only two 75 mg. doses of Demerol®. Her peristalsis returned in about 12 hours and she had no difficulty with urination. She commented on how pleasant her hospital visit had been and how relaxed she had felt.

Summary

The above five cases have been briefly summarized to illustrate not only how effective hypnotherapy can be in general practice but also to refute the widely held belief that this modality is time consuming. These cases also show that direct symptom removal while in light trance can be extremely effective and makes possible a marked reduction or elimination of analgesics and/or tranquilizers.

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Clinical Incidence of Renal Amyloidosis in Rheumatoid Arthritis

JOHN H. BLAND, M.D.*

INTRODUCTION

Amyloidosis is common in rheumatoid arthritis and when it occurs, renal involvement is usual. The incidence of renal amyloidosis in rheumatoid arthritis as determined by postmortem examination ranges from 26% to 61% as reported by Calkins and Cohen[3] and by Teilum and Lindahl[17] respectively. Unger, et al[20] studied 58 autopsied cases of patients with rheumatoid arthritis; there were 8 with renal amyloidosis; in no case was amyloidosis suspected antemortem; they also studied 56 clinical cases of rheumatoid arthritis and noted 6 cases of renal amyloidosis for an incidence of 9%; proteinuria was present in all these cases. Pollak, et al[10] in a study by renal biopsy of 41 patients with rheumatoid arthritis, discovered 4 cases of amyloidosis or a clinical incidence of 10%; all these cases had proteinuria. Sinclair and Cruickshank[12] reported 4 cases of renal amyloidosis in 16 postmortem examinations of patients with rheumatoid arthritis.

The high incidence of renal amyloidosis in post mortem studies of rheumatoid arthritis suggested two possibilities:

1. that amyloidosis and the pathogenetic abnormality in protein metabolism that results in amyloidosis may be integral to and a part of the pathologic process of rheumatoid arthritis; it is at least an unexplained frequent association.[12]
2. that since the incidence is so high postmortem, it might well be detected much earlier by more intense study for amyloidosis in patients with clinical rheumatoid arthritis. The postmortem cases of rheumatoid arthritis and amyloidosis presumably had more fully developed disease than might be expected in early development of amyloidosis in clinical cases of rheumatoid arthritis.

In a series of clinical studies of renal function in patients with rheumatoid arthritis, Sorensen[14,15,16] compared endogenous creatinine clearances in patients with rheumatoid arthritis and normal controls and found a very significant decrease in glomerular filtration rate in the rheumatoid patients; the low GFR was positively correlated with duration of disease and with seropositivity (positive rheumatoid factor tests). Possible explanation suggested to explain low GFR in rheumatoid arthritis patients were renal amyloidosis, infectious kidney diseases, unidentified specific rheumatoid renal lesion or toxic effects of drugs.

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Fearnly and Lackner[7] found 8 cases of amyloidosis in a one year study period among 183 patients with known rheumatoid arthritis. The clinical pointer to the presence of renal amyloidosis was unexplained proteinuria noted on routine urinalysis. Solomon[13] reported a case of a 36-year-old man with a 6 year history of rheumatoid arthritis who developed proteinuria 8 months before his death; postmortem examination disclosed renal and splenic amyloidosis.

Nagy et al[9] observed renal glomerular lesions in biopsy specimen from 5 patients with known rheumatoid arthritis; none had amyloidosis.

These quoted data all suggest that there may be a connective tissue renal lesion intrinsic to the pathology of rheumatoid arthritis, a kind of "renal arthritis." Perhaps amyloid protein synthesis and deposition in glomerular capillary basement membrane may be integral to rheumatoid arthritis. When amyloid is histologically identifiable, it is probably far advanced and it is reasonable to hypothesize that the process begins at some point as a primary abnormality in protein metabolism and progresses gradually and subtly.

The study reported was done to determine the clinical incidence of amyloidosis in a large number of patients with definite or classic rheumatoid arthritis as defined by the American Rheumatism Association criteria.[1] Simple clinical laboratory and biopsy techniques were used. Ninety-five to 98% of patients with proven renal

Age Distribution of 192 Patients With Rheumatoid Arthritis

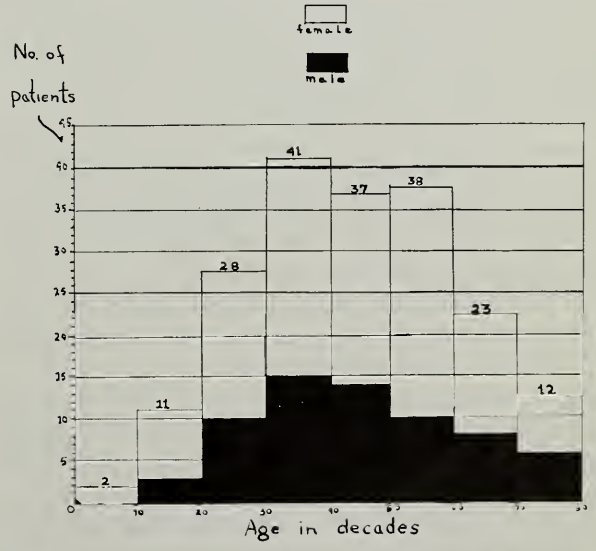


Fig. 1. Solid blocks represent males and open blocks females.

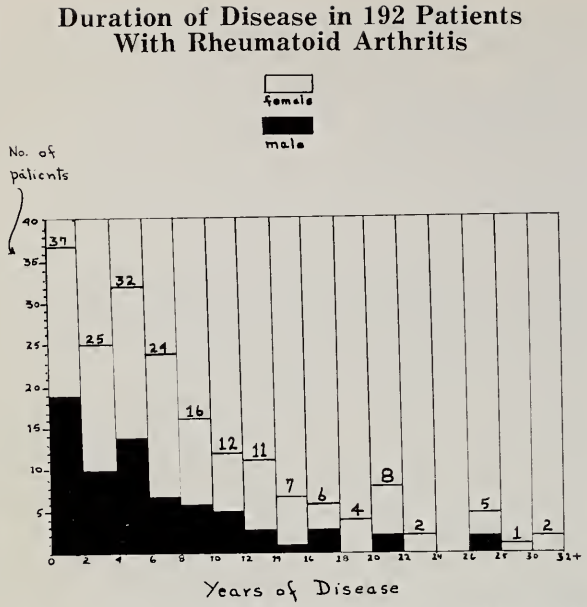


Fig. 2. Solid blocks represent males and open blocks females.

Total No. Patients	Sex M	Sex F	Duration of Disease	No. of Patients with Proteinuria	Proteinuria >0.25 gms for 24 hrs	Abnormal 15 min. PSP Test	Positive Gum biopsy	Latex Positive
192	66	126	1-32 yrs	40	19	4	9	135 70%

Fig. 3. Survey of 192 patients with Rheumatoid Arthritis.

amyloidosis have proteinuria; therefore the screening test was that of simple repeated qualitative tests for proteinuria (sulfo-salicylic acid test); if proteinuria was repeatedly noted further laboratory and tissue biopsy studies were done.

Methods

The study was started in 1956 and continued to 1960. Patients were selected for inclusion if they fulfilled the ARA criteria for definite or classic rheumatoid arthritis.[1] They were referred to the University of Vermont Rheumatism Research Unit and were drawn from clinics or from patients at the Mary Fletcher Hospital. A total of 192 patients were included in the study. Their age and sex distribution is given in Fig. 1. Fig 2 depicts their duration of disease. No particular effort was made to select patients according to duration of disease.

If they had 3 casual urine tests positive for protein, they had the following studies done:

1. Two consecutive 24 hour urine collections were made, volume determination and a quantitative protein determination made.

Fig. 3 summarizes the clinical data in this study. Note the incidence of positive latex test was 135 or 70%. Fig. 4 summarizes the clinical data in the 9 patients with gum biopsies positive for amyloidosis. Their age range was 29 to 74 years; duration of rheumatoid arthritis was 2 to 28 years; the serum latex was positive in 4 patients and negative in 5 patients.

	Sex	Duration of Disease	Latex Test	Gum Biopsy
Eko	M 42	2 years	Positive	+
EB	F 29	11 "	Negative	+
HH	F 43	6 "	"	+
MA	F 60	16 "	"	+
RM	F 60	28 "	"	+
VC	F 46	6 "	"	+
DK	F 44	6 "	Positive	+
HL	M 55	4 "	"	+
BF	F 74	14 "	"	+

Fig. 4. Cases of Amyloidosis and Rheumatoid Arthritis.

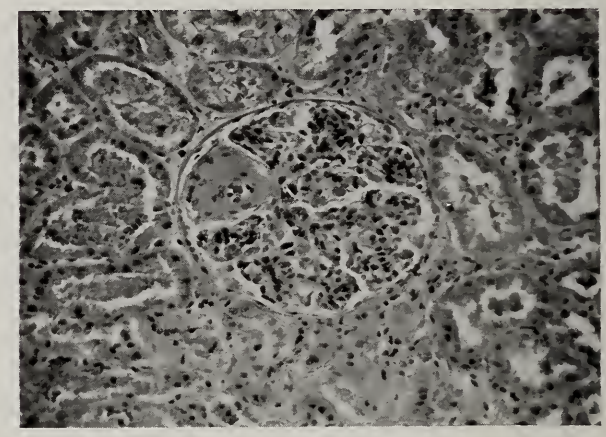


Fig. 5A

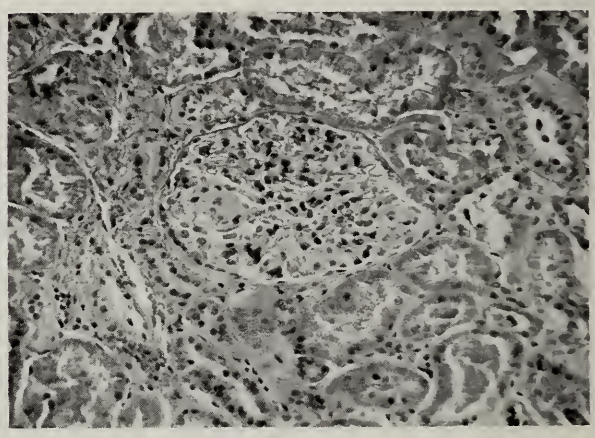


Fig. 5B

Figs. 5A and B. Note amyloid deposit in glomerulus.

Discussion

Out of a survey of 192 patients with definite or classic rheumatoid arthritis 40 had proteinuria by qualitative tests at least 3 times; 19 of the 40 excreted more than 0.25 gm. of protein per 24 hours on at least two 24 hour urine specimens; 4 had abnormal 15 minute PSP tests; and 9 had positive gum biopsies for amyloidosis. Only 2 of these patients had clinically suspected amyloidosis. We suspect that the clinical incidence of amyloidosis may be considerably higher than noted here. Asymptomatic amyloidosis has been reported by Levine, et al.[8]

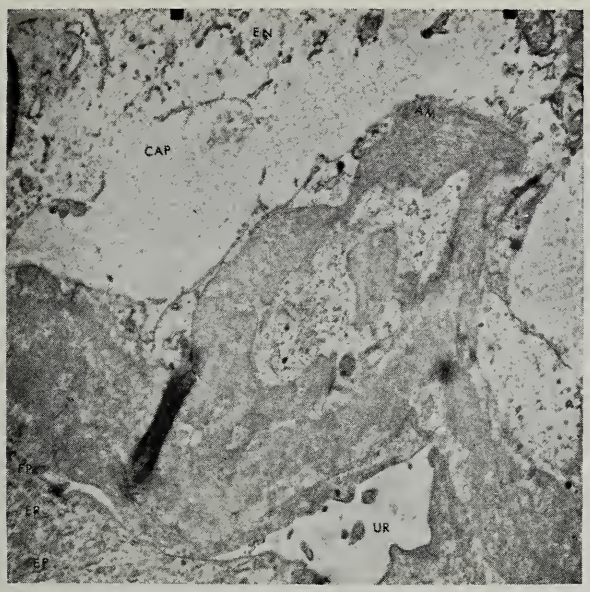


Fig. 6A. Shows capillary space (CAP), urine space (UR), and amyloid material (AM).



Fig. 6B. Further magnification of the same tissue (Fig. 6A) showing clearly the fibrillar amyloid in the basement membrane.

We suggest that repeated qualitative tests for proteinuria of patients with rheumatoid arthritis serves as a clinical detection method for the presence of amyloidosis. When 3 or more qualitative tests are positive a more extensive study entailing quantitative 24 hour urine, protein electrophoretic fractionation, and rectal mucosa, gum mucosa, kidney or liver biopsy are indicated. Though there is no satisfactory treatment of amyloidosis and only a little evidence exists suggesting it may resorb,[11] it is important that it be recognized as early as possible in rheumatoid arthritis since it is one of the few potentially fatal complications of rheumatoid arthritis.

Since amyloidosis is in rather high incidence in postmortem studies of rheumatoid arthritis and is frequently detectable in living patients, the possibility that it may be integral to the pathogenesis of rheumatoid arthritis is suggested. Amyloid has the chemical characteristic of a glyco-protein,[3,4,5] an immunoglobulin, but has recently been shown to be fibrillar morphologically in electron microscope studies.[19,2,6] We have been able to confirm this in some further studies of renal biopsy material by electron microscopy.

Fig. 5, A and B, are histologic sections of renal tissue from one of our patients studied by light microscopy.

Fig. 6, A and B, are electron microscope sections from the kidney of the same patient showing the fibrillar nature of the amyloid material in the basement membrane.

Conclusion

1. In a survey of 192 patients with definite or classic rheumatoid arthritis, 40 were found to have positive qualitative tests for protein in their urine; 19 were excreting more than 0.25 gm/24 hours and 9 had histologic evidence of amyloidosis in gum tissue biopsies.

2. Several patients had no symptoms related to amyloidosis. The possibility that amyloidosis in rheumatoid arthritis may be integral to the pathogenesis of that disease is suggested.

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Report of AMA Delegate

Paul H. Pfeiffer, M.D.

A special session of the House of Delegates of the AMA met in Chicago at the beginning of this month to discuss the effects of Public Law 89-97. This is the Health Insurance for the Aged and Medical Assistance Act signed into law by President Johnson last July in Independence, Missouri in the presence of former President Truman.

The day before the delegates met the AMA arranged for a panel to brief us. On this panel were Secretary of Health, Education and Welfare, John Gardner, Arthur Hess and Dr. Phillip Lee of the Social Security Administration and representatives of the Blue Cross, Blue Shield and American Hospital Association. The representatives of the government and "the Blues" were most cooperative. They were unable to completely answer all questions fired at them since many of the details of administering the new law remain in the discussion phase. They appeared willing to work with the Board of Trustees of the AMA and other AMA committees and have already modified some of the legislation in response to pressures from organized medicine. The delegates were divided in their reaction to the government. Some felt as do Dr. Appel and the present AMA leadership that every effort must be made to keep channels of communication open and maintain a "dialogue" between the Federal government and the medical profession and do whatever possible to mitigate the "medicare laws" and make them as consistent as possible with good medical care. They point out that one of the reasons the National Health Plan in England is so bad is that the British Medical Association refused to communicate with the government when the Plan was being drawn up. Others felt that the government leaders could not be trusted, that the nation has been "sold down the river" by the politicians and their labor backers and that the medical profession should do what it can to scuttle medicare and make it unworkable. A previous AMA President, Dr. Edward Annis, championed this view in most dramatic terms and an enthusiatic and eloquent band of delegates followed his lead.

I would like to be able to dissect the law for the benefit of the physicians of Maine. However, the language is such as to make my head swim. The following is an example taken from page 119 the third paragraph: "(5) If a widower, after attaining the age of 62, marries an individual (other than one described in subparagraph (A) or (B) of paragraph (4), such marriage shall, for purposes of paragraph (1), be deemed not to have occurred: except that, not withstanding the provisions of paragraph (3) and subsection (q), such widower's insurance benefit for the month in which such marriage occurs and each month thereafter prior to the month in which the wife dies or such marriage is otherwise terminated, shall be equal to 50 per centum of the primary insurance amount of the deceased individual on whose wages and self-employment income such benefit is based."

The AMA gave us an analysis which was better but still confusing. An excerpt of the analysis follows: "The amount payable for inpatient hospital services furnished an individual during a spell of illness will be reduced by an inpatient hospital deductible, which until 1969 will be $40, or if less, the charges imposed on the individual by the hospital for the services. However, if the customary charges for the services are greater than the charges imposed, the customary charges would be considered the charges imposed. The amount of payment will be further reduced by a coinsurance amount equal to one-fourth the inpatient hospital deductible for each day (before the 91st day) after the individual has been an inpatient for 60 days during that spell of illness." If they are not already doing so it seems high time the medical schools started courses in the interpretation of legalese jargon.

During the House of Delegates debate there were two issues which attracted most of the attention. (1) The non-participation issue as related to acceptance of fees and so forth. Legal counsel for the AMA was quite firm in stating that a medical society would be held liable under the Sherman Act relating to restraint of trade if it coerced its members into non-participating in a law of the land which might cause individuals to be deprived of services to which they were entitled. However, each physician acting as an individual may decide for himself whether or not he will participate. (2) There was much discussion over the makeup of the utilization committees which are a necessary part of the law. Most of the delegates felt that these committees should be made up of practising physicians and a resolution to this effect was overwhelmingly approved.

Under Part B — Supplementary Medical Insurance Benefits for the Aged — payments to the physician are to be made by an intermediary which may be a Blue Shield Plan, a local Medical Society or a private insurance carrier. Instead of a national fee schedule payments will be made on the basis of "reasonable charge for services . . . there shall be taken into consideration the customary charges for similar services generally made by the physician or other person furnishing such services, as well as the prevailing charges in the locality for similar services." There are two ways in which a physician can be reimbursed. He can charge and collect his usual fee from the patient who can then present the itemized, receipted bill to the intermediary and try to collect from them. Or else the physician can take an assignment from the patient, in which case he accepts 80% of the set fee from the intermediary (after the patient has payed the $50 deductible) and then can collect the remaining 20% from the patient or a local welfare agency. It was pointed out that the physician has the right to vary the method of payment from one patient to the next as he sees fit. Disregarding for the moment the ethical considerations of participation versus non-participation, Medical World News points out in the October 15, 1965 issue that the physicians of Maine (excluding the pediatricians and obstetri-

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State Of Maine

Department of Health and Welfare

DEAN H. FISHER, M.D.
COMMISSIONER

Public Assistance Medical Care in Maine Fiscal Year Ended June 30, 1964

EDSON K. LABRACK, M.P.H.*

Expenditures for medical care services provided for recipients of public assistance during the fiscal year ended June 30, 1964 amounted to $5,964,392. The was 26.8% of all expenditures for public assistance during the period. Money amounts shown in this report are expenditures for services actually provided during the fiscal period, hence they will vary slightly from fiscal reports of the Department which represent bills paid during the period.

During the fiscal year 1964 expenditures were up by about $422,000, or 7.6% over expenditures for 1963 and by $1,850,000 or 45.3% over 1962 which was the first year for the MAA program.

While expenditures increased in 1964 the number of persons provided with medical care actually decreased. In 1964 there were 47,550 persons in the State who were recipients of public assistance. This represented 48.1 public assistance recipients per 1,000 residents of the State. Medical care was provided for 11,301 of these recipients. The recipient rate for medical care among public assistance recipients was 237.7 per 1,000. Both the number of recipients and the recipient rate for medical care declined slightly between 1963 and 1964. In fiscal 1964 there were 11,578 recipients of medical care and the recipient rate was 239.1 per 1,000 recipients. The decline can be attributed to a general decline in the number of recipients on AABD and AFDC programs. See Table 2.

The phenomenon of increasing expenditures for a declining number of persons can be explained by an increase in the volume of services for MAA and AABD patients, and an increase in the number of MAA patients. The mean expenditure per person receiving medical care services increased between 1963 and 1964 on all programs. See Table 2.

The largest expenditure was for nursing home care. Nursing home care is provided for recipients on public assistance income maintenance programs, but not for MAA recipients. Nursing home care was provided for 2,922 recipients at a cost of $3,174,024, or 53.2% of all expenditures for medical care services. Hospital care is provided on all programs. There were 8,946 recipients of hospital care and expenditures for hospitalization amounted to $2,788,691, or 46.6% of all expenditures. Other types of medical care services including comprehensive medical clinic, visiting nurse services, prescribed drugs, physician office visits, dental care, and other services were provided on a limited basis for MAA recipients and selected recipients of rehabilitative services. Expenditures for these services amounted to $11,677 or 0.2% of all medical expenditures.

TABLE 1

TOTAL EXPENDITURES FOR PUBLIC ASSISTANCE MEDICAL CARE AND NUMBER OF PERSONS SERVED BY PROGRAM, MAINE, FISCAL YEARS 1963 AND 1964.

Program	1964		1963		Per cent change 1963-1964	
	Persons	Expenditures	Persons	Expenditures	Pers.	Exp.
Total	11,301	$5,964,392	11,578	$5,542,670	−2.4	+7.6
AABD	5,575	4,297,025	5,821	4,091,036	−4.2	+5.0
AFDC	2,116	364,849	2,479	370,199	−14.6	−1.4
MAA	3,610	1,302,518	3,278	1,081,435	+10.1	+20.4

TABLE 2

RECIPIENT RATES AND AVERAGE EXPENDITURES PER PERSON RECEIVING PUBLIC ASSISTANCE MEDICAL CARE SERVICES, BY PROGRAM: MAINE, FISCAL YEARS 1963 AND 1964.

Program	Medical care recipient rate per 1,000 PA recipients		Mean expenditure per recipient served	
	1964	1963	1964	1963
Total	237.7	239.1	$527.78	$479.58
AABD				
Aged	361.1	365.9	802.39	740.09
Blind	231.8	288.9	529.11	454.87
Disabled	291.1	320.7	620.29	539.73
AFDC				
Children	49.4	58.7	154.54	125.10
Adults	151.1	160.4	188.85	173.75
MAA	1/	1/	360.81	329.91

1/ Not computed

*Director, Division of Research and Vital Records

TABLE 3

EXPENDITURES FOR PUBLIC ASSISTANCE MEDICAL CARE SERVICES AND NUMBER OF RECIPIENTS, BY KIND OF SERVICE: MAINE, FISCAL YEARS 1964 AND 1963.

Kind of Service	Number of recipients		Expenditures	
	1964	1963	1964	1963
Total	11,301	11,578	$5,964,392	$5,542,670
Hospitalization	8,946	9,124	2,778,691	2,563,216
Nursing home care	2,920	2,925	3,174,024	2,880,085
Other medical care	131	132	11,677	7,493

Hospital care is provided for recipients of public assistance on all programs. During the fiscal year 1964 hospital care was provided for 8,946 persons and expenditures amounted to $2,778,691. The number of persons hospitalized declined slightly between 1963 and 1964 but expenditures for hospital services increased by slightly over $200,000. Analysis of expenditures for hospitalization indicates that nearly all of the increased expenditure can be attributed to expansion of MAA program. Expenditures for hospitalization of MAA recipients was up by $222,432 in fiscal 1964. Expenditures for hospitalization declined for aged and disabled recipients of AFDC.

The number of hospital days for public assistance recipients discharged from general hospitals in fiscal 1964 was 151,860. This was about 7.7% of all hospital days utilized in the State during the year. There were 12,100 hospital discharges of recipients during the year. The average duration of a hospital episode was 12.5 days. The average duration of hospitalization in 1964 was 1.1 days longer than in 1963. The increased average duration was apparent for all types of recipients except child recipients of AFDC. Table 4 shows data for hospitalization in 1964.

Reports of hospital discharges of recipients provide information as to the "primary" reason for hospitalization as determined at time of discharge. Although a complete analysis of morbidity data is beyond the scope of this report, certain summary data may be appropriate. Disease of the digestive system represented the most common reason for hospitalization of recipients, diseases of the heart was second and diseases of the respiratory system was third in order of importance. Together these three categories accounted for about 40% of all hospitalizations. As might be expected, the primary diagnosis varied from program to program. Aged and disabled persons on AABD program showed similar patterns with the most common causes as listed above, although diseases of the heart were more frequent in disabled than in aged recipients. Neoplasms was the third most common diagnosis for MAA recipients while it ranked 5th for OAA recipients. For adult recipients of AFDC about 30% of all hospitalizations were related to pregnancy and delivery, while 34.4% of AFDC children were hospitalized for "Upper respiratory diseases" (mostly tonsils and adenoids).

The duration of hospitalization for particular conditions ranged from a high mean duration of 20.1 days for vascular lesions of the central nervous system to a low mean of 4.4 days for hospitalization related to pregnancy and delivery.

TABLE 4

HOSPITAL SERVICES FOR PUBLIC ASSISTANCE RECIPIENTS BY PROGRAM: MAINE, FISCAL YEAR 1964.

Program	Number hospitalized	Expenditure	Number of days	Number of discharges	Mean days per hospitalization
Total	8,946	$2,778,691	151,860	12,100	12.6
AABD					
Aged	2,623	913,407	48,391	3,583	13.5
Blind	80	20,652	1,065	98	10.9
Disabled	573	188,774	10,225	842	12.1
AFDC					
Children	1,013	156,545	7,952	1,368	5.8
Adult	1,102	203,666	9,904	1,491	6.6
MAA	3,555	1,295,647	74,323	4,718	15.8

TABLE 5

PUBLIC ASSISTANCE RECIPIENTS OF NURSING HOME CARE BY DURATION OF CASE AND PROGRAM: MAINE, FISCAL YEAR 1964.

Months of care	AABD Total	Program: Aged	Program: Blind	Program: Disabled
Total	2,920	2,953	30	297
Less than 1	235	216	2	17
1 less than 2	427	363	5	59
2 less than 3	192	169	2	21
3 less than 4	184	167	—	17
4 less than 5	110	96	2	12
5 less than 6	131	113	6	12
6 less than 7	97	92	—	5
7 less than 8	108	100	1	7
8 less than 9	116	103	2	11
9 less than 10	96	83	1	12
10 less than 11	138	113	1	24
11 less than 12	240	203	3	34
12	846	775	5	66

Nursing home care was provided for 2,920 recipients of AABD. Total expenditures for nursing home care provided during the fiscal year 1964 amounted to $3,174,024. Over 50% of all nursing home beds in the State were occupied by public assistance recipients. In total, the Department provided over 19,000 months of nursing home care for recipients. While the number of public assistance patients in nursing homes did not change between 1963 and 1964, expenditures for nursing home care were up by nearly $300,000. The increase in expenditures was due to an increase of over 2,000 in the number of months of nursing home care. Payments for nursing home care were increased to $200 per month in May 1964.

In general, public assistance patients in nursing homes tend to be long term patients. In 1964 there were 846, or 28.9% of all patients, who were in the home for the entire year. This was an increase over the number in 1963 which was 581, or 19.9%. Table 5 contains data on number of months of care in nursing homes in 1964.

Other medical care services were provided on a limited basis.

Maine Heart Association Notes

116 State Street, Augusta, Maine

Diabetes Mellitus and Atherosclerosis

"The detailed analysis of the experience at the Joslin Clinic - - - provides important information regarding the inter-relation of acute myocardial infarction and diabetes mellitus. - - -

"The baleful effect of diabetes in promoting atherosclerosis of the coronary and other arteries is disclosed by the aspects of acute myocardial infarction in diabetic patients that differ from the experience generally observed in nondiabetic persons. - - - in the presence of diabetes women either equal or outnumber men at every age. The increasing incidence of acute myocardial infarction with the increased duration of diabetes and the frequency of advanced atherosclerosis observed in youthful diabetic patients emphasize the grave vascular influence of diabetes mellitus. - - - these patients not only have more severe atherosclerosis but acquire atherosclerotic lesions ten to twelve years earlier than nondiabetic persons.

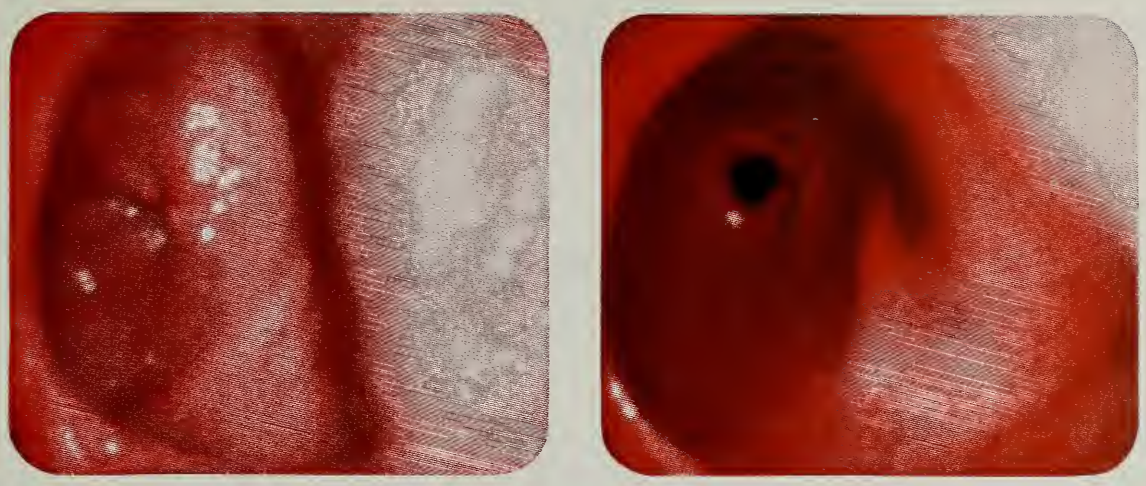
"The exact nature of the mechanisms that promote the development of atherosclerosis in diabetic patients has been the subject of much conjecture. - - - The evidence to date strongly indicates some unidentified factors inherent in the diabetic state that have up to now eluded all investigators.

"- - - any diabetic person of either sex over the age of forty can be assumed to have severe coronary sclerosis, especially if he has had diabetes for ten years, or if he has hypertension in addition to diabetes. In the presence of a compromised coronary arterial circulation anoxia, fall in blood pressure or anemia due to blood loss during operation often precipitates acute myocardial infarction. The special precautions in preoperative, operative and postoperative management - - -, particularly in diabetic patients, are clear.

"The importance of diabetes in relation to atherosclerosis - - - indicates the advisability of testing for diabetes by glucose tolerance tests or blood sugar determinations after meals in many patients with angina pectoris, acute myocardial infarction or peripheral vascular disease who may not exhibit glycosuria under usual circumstances."

Submitted by Jacob B. Dana, M.D.

Reference: Editorial. The New England Journal of Medicine, Vol. 273, No. 9, August 26, 1965, pages 505-506.



Intragastric photography studies[1]

A/ E. B., male, age 48. Normal antral contraction. Pyloric opening is not seen. It is difficult to differentiate a deep prepyloric contraction from a "pyloric fleurette" or true pylorus.

B/ Same subject after 6 mg. of propantheline bromide intravenously; antral contractions ceased. The pyloric orifice remained open and was easily identified. Better visualization of the antrum was also obtained.

Now you can see Pro-Banthine® at work
(propantheline bromide)

Pro-Banthine is so effective in anticholinergic action that it may be employed in visualizing the entire pyloric region.

In addition to the intragastric photographs, cinegastroscopic studies[2] have demonstrated graphically not only its effectiveness but the superiority of Pro-Banthine over belladonna alkaloids.

Pro-Banthine produced complete cessation of gastric, antral and pyloric motor activity with a dose of 6 mg. intravenously. This is approximately one-third the usual oral dose of 15 mg.

Atropine at full normal dosages did not produce such cessation. It required double the usual oral dose of atropine, 0.8 mg. intravenously, to duplicate the aperistaltic action of Pro-Banthine. This dose of atropine produced pronounced discomfort and tachycardia with ventricular rates as high as 150 per minute.

It is this pharmacologic superiority of Pro-Banthine which has made it the most widely prescribed anticholinergic in such conditions as peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Dosage—The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six 15 mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications—Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. The drug is contraindicated in patients with glaucoma or severe cardiac disease.

1. Barowsky, H.; Greene, L., and Bennett, R.: Investigators' Clinical Report. Photographs courtesy of Drs. H. Barowsky, L. Greene and R. Bennett.

2. Barowsky, H.; Greene, L., and Paulo, D.: Paper read at Meeting of American Society for Gastrointestinal Endoscopy, Montreal, Canada, May 25-27, 1965.

SEARLE

Research in the Service of Medicine

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

ANORECTAL CANCER

Henry Finch, M.D., *Atlanta*

PHYSICIANS! PATIENTS! Don't pay any attention to that severe chest pain! It may be nothing serious or it may be due to a heart attack. PHYSICIANS! PATIENTS! Don't pay any attention to rectal bleeding! It may be nothing serious or it may be due to a cancer of the anorectal sigmoid area.

77.2 per cent of all intestinal cancers are in the anorectal area. Carcinoma of the anorectal sigmoid area represents the most frequent form of internal malignancy. It is readily diagnosable by inspection, palpation, anosigmoidoscopy and X-ray. It is estimated, based on over 10,000 cases reported in the literature, that 80.4 per cent of all intestinal cancers can be seen through sigmoidoscope. Radical surgery with early carcinoma of the rectum and colon gives the highest cure rate of any internal cancer in the body.

Symptoms

Bleeding either bright or dark blood is the most frequent and outstanding symptom of cancer of the lower colon. Every patient who presents himself with rectal bleeding should, therefore, have an anosigmoidoscopic examination before any local therapy is instituted. Any change in bowel habit, a feeling of urgency, incomplete evacuation, and pain in the lower rectum and back are some of the other symptoms that demand an anosigmoidoscopic examination.

Excellent results can be obtained in the management of carcinoma of colon and rectum if the early detection through routine anosigmoidoscopic examination is performed. Colon resection abdominoperineal or "Pullthru" are the treatments of choice.

Polypoid Growths

These may be single or multiple, sessill or peduncutated adenomatous growths of the mucous membrane. Various reports place incidence of polypoid growths found on routine anosigmoidoscopic exam from two to 11 per cent. When they are found, a barium enema with air contrast studies becomes mandatory. How many of these polyps become malignant is uncertain, but it is certain that a polyp that has been removed cannot become cancerous. When these polyps are found with the sigmoidoscope they should be removed through the sigmoidoscope. When polyps are found with barium enema, they should be removed transabdominally.

If every physical will include inspection, palpation and anosigmoidoscopic examination, instead of more people dying from anorectal colon cancer than any other cancer, more people will be cured than from any other internal cancer in the body.

490 Peachtree Street N.E.

Approved by the Professional Education Committee, Geargia Division, ACS.
Reprinted from The Journal of the Medical Association of Georgia, December, 1964, Vol. 53, Page 393.

Necrology

Arthur N. Lieberman, M.D.

1914-1965

Arthur N. Lieberman, M.D., 51, of Bangor, Maine died on September 27, 1965. He was born in Bangor, Maine on January 20, 1914, son of Abraham and Minnie Lieberman.

Dr. Lieberman graduated from Bowdoin College in 1935, studied a year at the University of Maine and became associated with the Roscoe B. Jackson Memorial Laboratory in Bar Harbor. He received his medical degree from the University of Michigan Medical School in 1942. He interned at the Kings County Hospital in New York from 1942 to 1943 and served in the U. S. Army Medical Corps from 1943 to 1946, including two years in the European Theatre. He was discharged as a Major in the Medical Corps. In December, 1946 he set up his practice in Bangor, where he was a member of the staffs of the Eastern Maine General Hospital and St. Joseph Hospital, and where he practiced until his death.

Dr. Lieberman was a member of the Maine Medical Association, the Penobscot County Medical Association, the American Medical Association and was a former president of the Bangor Medical Club. He was also a member of the Beth Abraham Synagogue, a board member of the Jewish Community Council and the Bangor Hebrew School.

Surviving besides his parents, are his wife, Florence (Kaminsky) of Bangor; a daughter, Miss Diane Wendy Lieberman of Bangor; two brothers, Max of Boston, Massachusetts and Leo of New York City; several nieces and nephews.

County Society Notes

KENNEBEC

The Kennebec County Medical Association met on September 16, 1965 at the Holiday Inn in Augusta, Maine.

The meeting was called to order by the President, John D. Denison, M.D. Dr. Denison, as Councilor for the Fourth District of the Maine Medical Association, presented a report of the recent meeting of the Council of the Maine Medical Association. It was noted that approximately 200 members had contributed this year to the Maine Medical Education Foundation and that many more contributions were needed.

Theodore C. Barton, M.D., instructor in obstetrics and gynecology at Harvard Medical School and assistant obstetrician at the Boston Lying-In Hospital, was the speaker of the evening. He spoke of the attempts at lowering fetal mortality in erythroblastosis by intraperitoneal blood transfusions of the fetus, describing in some detail the methods of following the condition of the erythroblastotic fetus and the method of the transfusion itself. He also discussed induction of labor techniques in cases of fetal death.

Earle M. Davis, M.D.
Secretary

LINCOLN-SAGADAHOC

Seventeen members were present at the meeting of the Lincoln-Sagadahoc County Medical Society which was held at The Ledges Inn in Wiscasset, Maine on September 21, 1965.

The meeting was called to order by the President, Carl R. Griffin, Jr., M.D. A letter was read from the Boothbay Regional High School thanking the society for the subscription to "Today's Health."

Hamdi Akar, M.D. accepted the appointment as chairman of the Diabetes Committee of the society.

A letter from the State Department of Health and Welfare was read explaining a counseling and referral program for Armed Forces Medical Rejectees. John F. Dougherty, M.D. spoke on the necessity of cooperation of each member of the society in this program.

The annual election of county society delegates and alternates to the Maine Medical Association House of Delegates will be held at the October meeting.

Alan W. Zeller, M.D. proposed that costs of society meetings be borne by the entire membership. It was voted unanimously that as of January 1, 1966 the society dues shall be raised to $50.00 annually; all meeting expenses, including meals, are to be paid from society funds.

Dr. Dougherty discussed the non-discrimination statements which are to be filed with the State Department of Health and Welfare and stressed the need for filing these forms.

S. J. Bullington, M.D., the guest speaker, gave an illustrated talk about eye problems in general practice.

George W. Bostwick, M.D.
Secretary

ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at the Central Maine General Hospital in Lewiston, Maine on September 16, 1965.

The meeting was called to order by the President, Henry C. Thacher, M.D. It was voted to approve the formation of the Androscoggin County Section of the American Association for Medical Assistants. It was voted to approve the measles clinic being sponsored jointly by the Lewiston Jaycees and the Health Department.

Dr. Thacher read a proposal to implement the formation of a Public Health Committee. No such committee has been appointed although mentioned in the Bylaws. It was voted that this proposal be properly phrased, have perusal and approval of councilors and be submitted at the next meeting to be placed in proper place in the Constitution.

There was a panel discussion concerning difficulties arising between doctors and pharmacists and it was agreed that a committee might iron out existing problems.

Donald L. Anderson, M.D.
Secretary

HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on October 13, 1965.

Thomas H. Palmer, Jr., M.D. of Bangor, Maine, the guest speaker, spoke on the recognition and management of blunt and sharp penetrating wounds of the abdomen.

Russell G. Williamson, M.D.
Secretary

REPORT OF THE AMA DELEGATE—*Continued from Page 255*

cians) stand to earn an average of $6,828 per man as a result of Medicare benefits.

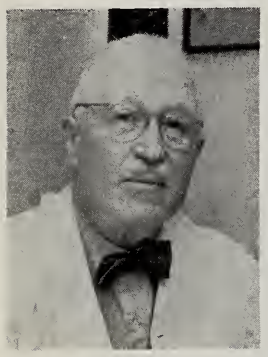
There was little discussion at this meeting of Part 2 under Title I of P.L. 89-97 which deals with federal grants to the States for medical assistance programs. This portion of the bill deals with Aid to Dependent Children and the needy of all ages. In the long run it may affect the practise of medicine more than the medicare provisions for the aged.

The AMA leadership has succeeded in modifying the so-called DeBakey Bill for the establishment of regional complexes to study and treat Heart Disease, Cancer and Stroke. Meeting with government officials in Washington, the AMA was able to reduce the appropriation by three-fourths and to establish that these medical centers could not be established unless there was local demand and that no patient could be referred there other than by a practising physician.

The AMA leadership deserves the support of the membership in its efforts to maintain high-quality medical care on a fee-for-service basis. For the moment, at least, there appears to be an amicable "dialogue" between the AMA and the federal government.

News and Notes

Huddilston Medal Awarded to Thomas A. Foster, M.D., of Portland



Thomas A. Foster, M. D., Portland Pediatrician, was awarded the Roselle W. Huddilston Medal for his "outstanding contributions in the general field of health to the people of the State of Maine." The Medal was presented by Frederick T. Hill, M.D., 1958 Medalist, during the annual meeting of the Maine TB and Health Association in Bangor, October 13, 1965.

Prior to retirement Dr. Foster was chief of Children's Medical Services at the Maine General Hospital (now Maine Medical Center), Portland. He was also Pediatric Consultant to the Knox County General Hospital, Rockland and was on the consulting staff of the Goodall Hospital, Sanford and St. Andrews Hospital, Boothbay Harbor.

One-time president of the Maine Pediatric Society, he was the first pediatrician in Maine to be certified by the American Board of Pediatrics. He is also a past president of the Maine Medical Association and is an honorary member.

Dr. Schumacher Elected National Group President



William E. Schumacher, M.D. of Augusta, Maine, director of Maine's Bureau of Mental Health, has been elected president of the National Association of State Mental Health Program Directors, according to a recent announcement by Harry B. Schnibbe, executive secretary of the association.

Walter F. Ulmer, commissioner of the Department of Mental Health and Corrections of Maine, said Dr. Schumacher's election to one of the key jobs in mental health work in the U. S. is "a great honor for Maine and a personal tribute to Dr. Schumacher."

Dr. Schumacher is a native of New Jersey. He was graduated from Dartmouth College in 1943 and received his medical degree from the University of Vermont College of Medicine in 1945.

Dr. Schumacher was named Maine director of Mental Health in the Maine Department of Mental Health and Corrections in September, 1960.

STATEMENT OF OWNERSHIP, MANAGEMENT AND CIRCULATION

(Act of October 23, 1962; Section 4369, Title 39, United States Code)

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7. Owner: (If owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 percent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a partnership or other unincorporated firm, its name and address, as well as that of each individual must be given.)

Maine Medical Association, P.O. Box 637, Brunswick, Maine 04011.

8. Known bondholders, mortgagees, and other security holders owning or holding 1 percent or more of total amount of bonds, mortgages or other securities. (If there are none, so state): None.
9. Paragraphs 7 and 8 include, in cases where the stockholder or security holder appears upon the books of the company as trustee or in any other fiduciary relation, the name of the person or corporation for whom such trustee is acting, also the statements in the two paragraphs show the affiant's full knowledge and belief as to the circumstances and conditions under which stockholders and security holders who do not appear upon the books of the company as trustees, hold stock and securities in a capacity other than that of a bona fide owner. Names and addresses of individuals who are stockholders of a corporation which itself is a stockholder or holder of bonds, mortgages or other securities of the publishing corporation have been included in paragraphs 7 and 8 when the interests of such individuals are equivalent to 1 percent or more of the total amount of the stock or securities of the publishing corporation.
10. This item must be completed for all publications except those which do not carry advertising other than the publisher's own and which are named in sections 132.231, 132.232, and 132.233, postal manual (Sections 4355a, 4355b, and 4356 of Title 39, United States Code.)

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E. Total Distribution (Sum of C and D)	1269	1277
F. Office Use, Left-over, Unaccounted, Spoiled after Printing	17	20
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I certify that the statements made by me above are correct and complete.

ESTHER M. KENNARD, Business Manager

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Results of a Questionnaire Submitted to Physicians' Children

Frederick C. Emery, M.D.*

When physicians gather, a topic of conversation which often appears is our inability to spend enough time with our families, our interrupted family plans because of practice demands, our supposed failure to meet the requirements of a model PTA-going, little-league-swinging, girl scout-camping parent, and the effect this might have on the psyche of our progeny. During such a session, the idea occurred that no one had ever asked the ones most concerned – our children – what they thought about it. This is an attempt to find out.

A questionnaire was produced and sent out to our children. To provide anonymity, the questions were printed with checkoff answers, as far as possible. They were asked not to sign the form, and a self-addressed envelope was supplied. The letters were received and opened by a third party, so that the postmarks could not be noted. When all the returns were in, the answers were examined. It was hoped that assuring anonymity to the children would produce sincere and honest answers, and this was certainly accomplished. I was impressed with the time, effort, and thought obvious in all the replies. The forms were given to as many physicians' children as could be reached regardless of age or sex. Six groups were represented, ages 10 to 15, 15 to 20, and 20 and over, in both sexes. The fathers were from all specialties and from general practice. There were 148 forms sent out and 108 returned from 65 girls and 43 boys.

As doctors, expending our time, energies, and emotions in trying to be good doctors, we often worry about our ability to live up to the popular conception of what a good father should be. We wonder if we are providing a good home life for our families, and if our children like living in a doctor's family.

This is an attempt to find out, and we thought it would be enlightening to let the ones most concerned – the doctor's children – have their say on the subject.

We would like you to fill out this questionnaire and mail it in the self-addressed envelope. Don't sign it, as we do not wish to know who you are. Thus you can be completely honest in your answers. No one will know who said what. Please give the questions some thought, and don't ask for help in answering them. If you don't understand a question, ask your parents to interpret it for you, but do your own answering.

You "older" children, who may be on your own and raising your own family, just replace the "do's" with "did's," and fire away.

When we have gathered all the answers, we should be able to tell how we stack up with other parents, and if we fall behind your expectations, how to help remedy the situation.

Questionnaire

Please Circle Answers Where Possible

1. Are you a male or female?
2. What is your age group? 10 to 15 15 to 20 20 on up
3. Do you like being a physician's child? yes no
4. Do you think there are disadvantages in being a doctor's child? yes no
   If yes, please list:
5. Are there advantages in being a doctor's child? yes no
   If yes, please list:
6. Are you helped in your school or social life by being a physician's child? yes no
7. Are you hindered in your school or social life by being a physician's child? yes no
   If yes, please list:
8. Would you like to be a doctor? yes no
   Why?
9. Would you like your own children to become doctors? yes no
10. Who provides most of the discipline in your home? father mother
11. Is this the way you prefer it? yes no

*Chief of Pediatric Service, Eastern Maine General Hospital, Bangor, Maine.

12. Who should make the rules, decide the allowances, make the decisions, etc., for the family? father mother
13. Is your father too strict too lax or about right in your discipline?
14. Do your prefer family planned vacations and activities? yes no
15. Do you prefer individual activities and vacations? yes no
16. Do you have many planned activities called off because your father is a doctor? yes no
    If yes, do you resent this? yes no
17. Does your father have enough time to spend with the family? yes no about right
18. Do you think your father takes enough interest in you and your activities? yes no
19. Do you resent the time your father has to spend with patients? yes no
20. Do you have much contact with your father's practice and patients? yes no
    Do you want to? yes no
21. Do you understand what your father does as a doctor? yes no
22. Would you like to know more about his work? yes no
23. Do you worry about family finances? yes no
24. Do you worry about your father's patients? yes no
25. Do you think your father has to attend too many medical meetings? yes no
26. Do you think your father misses more PTA meetings, school functions, church activities, etc., than a non-physician father? yes no
    If yes, do you resent this? yes no
    Do your friends tease you about it? yes no
27. Does your father seem to prefer one member of the family over another? yes no
    If yes, do you resent this? yes no
28. Does your father show enough love and affection for you? yes no
29. Does your father expect too much from you in school work, chores, behavior, etc.? yes no
30. When you have a problem, do you prefer to talk it over with your mother or your father?
31. Do you like to be alone with your father? yes no
    Why?
32. When you are alone with your father does talk and companionship come easily? yes no
33. Does your father praise you enough? too much? too little?
34. Do you think people outside your family expect more of you because you are a doctor's child? yes no
35. Would you like to change some of your father's habits and ways? yes no
    If yes, list:
36. Now in your own words would you like to sum up your opinion of your life in a doctor's family and your father as a parent?

As the project took shape, some thoughts were expressed that sub-grouping into children of the specialties might be helpful. Perhaps life in the families of those doctors not seeing private patients, not making house calls, etc., might be different. Perhaps there would be a difference in the families of those physicians having offices in their homes. Perhaps the questions should have been selected from the ideas of several doctors instead of one. However, it seemed that the questions as given would help determine: first, if being physicians created problems for our children directly related to our occupation; second, if we were worrying needlessly about the effect of our professional lives on our family lives; and third, if a problem existed, what might we do to help solve it.

Several interesting reactions were noted as the wheels started to roll. Some fathers seemed to shudder a little when asked if their children could be questioned, and then wistfully wished they could know the answers their family would give. Others offered the information that once the questions were answered and in the mail there seemed to be a lessening of mild tensions around the house. Perhaps we were providing a little mental carthar-sis for our children. If we proved nothing else, we established that the doctor's children in any given community have the world's greatest grapevine going for them! The word flew, and indignant requests for questionnaires were received from those youngsters inadvertently omitted, or omitted because of lack of forms, or lack of time to transmit them.

Results

Reading these answers has been a turbulant emotional experience. The answers have been so sincere, so well thought out; some pleading, some depressing, some plaintive, all prideful, and all from the heart, that it has been difficult to be objective about them. I expected differences in the answers of the age groups, but, in general, this was not so. Differences were expected in the answers of the males and females, and there were minor differences to be noted later, but the key questions were answered remarkably alike.

Of the 108 questionnaires returned, 103 liked being a doctor's child, 3 did not, and 2 were undecided. Sixty-five thought there were disadvantages in being a physician's child, 47 did not. Ninety-eight thought there were advantages, and 9 did not. Seventy-one thought they were helped in their school and social life, and 32 felt they were not. Only 14 thought they were hindered in their life outside the home by being the child of a physician, and 89 did not. Sixty-three would like to become doctors, and 41 said "no." Fifty-eight would like their own children to become physicians, 8 would not, and 38 stressed the importance of letting the children decide for themselves. Forty said that dad provided the discipline, and 39 said that mother did, while 27 indicated that both parents had a hand in it. Whichever way it was done, 79 thought it was right, and 15 did not. It was interesting that of these 15, eleven had said that mother provided the discipline. Fifty thought that father should make the rules and family decisions, and only 3 thought that mother should, but 54 thought this should be a joint effort. Ninety-two said that their father was about right in his amount of discipline, 11 said he was too strict, and 4 too lax. Sixty-eight favored family planned activities over individually planned, and 27 did not. Of these 27 most were in the 15-20 age group. As one put it, "Togetherness of more than one day produces complete disintegration."

I was pleased to find that the majority, 60 vs 45, did

not think they had too many activities called off because dad was a doctor, and that of the 45 who did, twenty-nine did not resent it in the least. Several said they took pride in their father's dedication to his work and in his being willing to respond to an emergency. Some felt it gave them a feeling of sharing in his work of helping others. It was a relief to note that 65 thought their father did have enough time to spend with the family. Thirty-nine did not think so. Almost 100%, 100 to be exact, said they did not resent the time their father had to spend with patients and away from the family. The majority said they did not have much contact with their father's practice and did not want to. Only 5 said they did not understand their father's work, but 74 would like to know more about it. Most of the children did not worry about family finances or dad's patients, but those who did were mostly in the 15 to 20 age groups. Seventy-seven thought physicians did not have to attend too many medical meetings, and 68 thought their fathers did miss more PTA meetings, social functions, etc., than did non-physician fathers, but only 8 resented the fact. To our credit, 86 said that dad did not prefer one member of the family over another, and 17 said that he did. However, three of these did not resent the preference because they were the favored one, and three said "mom" was the preferred one and "that was the way it should be." Ninety-seven thought they were shown enough love and affection by their father, 8 did not think so. Twenty-one said their father expected too much from them in school grades and behavior, 81 said that he did not.

Fathers were way down the list as confidants as 42 preferred to talk over problems with mother, 33 preferred both parents and only 13 went to dad alone. There were 12 who did not wish to confide with either parent, 8 preferring someone outside the family, and 4 working out their problems by themselves. Eighty-four liked to be alone with their father and 12 did not. Eight said that talk and companionship came easily with dad, and 21 said that it did not. Eighty-three thought they were praised enough, 16 too little, and 2 too much. Fifty-three thought people outside the family expected more of them because they were physician's children, and 48 did not. Thirty-nine wanted to change some of their habits, 66 did not.

There were some differences of opinions among the age groups and the sexes. As might be expected, more girls said they did not want to become doctors than did boys. The boys were about 50/50 on this and the girls were 2 to 1 against it. The majority of females preferred family planned activities and vacations, and the majority of males were in favor of individual plans. In both sexes, the 15-20 age group was heavily in favor of individuality. Only the girls resented the fact that their fathers missed more outside activities than non-physician fathers, none of the boys did. A majority of the girls thought that more was expected of doctors' children, and a majority of the boys did not. The group with the highest percentage of its members stating there were disadvantages in being a physician's child was the 15-20 females.

Some of the specific questions and answers were revealing. Here are some of the disadvantages in being a doctor's child:

Females:

1. "Doesn't pay enough attention to us when we are sick." (This was mentioned frequently.)
2. "Late for meals."
3. "Don't like to hear medical talk at meals." (Mentioned several times.)
4. "Friends don't want to come to the house because they are scared of father."
5. "Doctors' kids are not supposed to get sick."
6. "When on call at the hospital, father has a terrible disposition."
7. "Father worries too much about his patients and gets ugly and irritable."
8. "Telephone rings too much." "Can't use the phone enough." "Life centers around the telephone."
9. "Uncertain and interrupted plans, lack of routine."
10. "Not able to use the car enough."
11. "Restricted activity and enforced quiet when office is in the home." (Mentioned several times.)
12. "Don't see father enough." (Most frequent complaint.)
13. "People expect you to have medical knowledge when you really don't. Other doctor's worst offenders."
14. "Catered to by teachers, neighbors, friends and thus tend to become snobbish."
15. "Not able to live up to doctor-parent's reputation."

Males:

1. "Expected to know and give medical advice."
2. "Interrupted activities."
3. "Lack of time with the family." (Disadvantages 2 and 3 were the most frequently listed by the boys.)
4. "Having to try to live up to father's image."
5. "Other kids are jealous of you."
6. "Too much telephone ringing." "Have to answer the phone too much." "Can't use the phone enough."
7. "Impossible to establish own identity because of living in father's shadow."
8. "Can't use the car enough."

Here are some of the advantages in being a doctor's child:

Females:

1. "Financial security."
2. "Free medical care." (Several mentions.)
3. "Free veterinary service."
4. "Help with studies." (This was frequently cited.)
5. "Takes care of you when you are sick." (Advantages 4 and 5 were the most frequent.)
6. "Have more independence because father doesn't have time to supervise every detail of your life."
7. "Get to know more people."
8. "Association with other doctors' families."
9. "Intellectually and educationally stimulating family life." (This was the second most frequent listing.)
10. "Get to have lots of brothers and sisters."
11. "Helped to be a better mother and homemaker."
12. "Pride in father's profession."
13. "Learn to appreciate good health and habits."

Males:

1. "Free medical care."
2. "Educated parents and educational home atmosphere." (This was most often mentioned.)
3. "Get to know lots of people."
4. "Pride in father's work, ideals, professional competence." (Noted many times.)
5. "Financial security."
6. "High social standing in the community."
7. "Father has a physically safe job."
8. "Gives one a better idea about good health."

The few who thought they had been hindered in their school and social life by being a physician's child gave these reasons:

1. "Expected to be like father by other people and usually disappointed them."
2. "Other kids showed envy of you."
3. "Teachers expected too much from you."
4. "Others think you are rich."
5. "Less easy to make friends."
6. "Expected to know and pass on medical knowledge."
7. "Adults try to pry into medical affairs of father's patients."

These are some of the reasons given why they liked to be alone with father:

1. "Stimulating companionship."
2. "Learning through conversation and absorbing father's knowledge." (This was the most frequent listing.)
3. "Gives more comfort than mother."
4. "He understands me, treats me as an equal, and respects my opinion." (This was the second most frequent listing.)
5. "Same interests."
6. "I love him."

The reasons why some children did not like to be alone with their father were:

1. "He makes me nervous and uncomfortable." (This was the most frequent complaint.)
2. "Always tells me my way is wrong."
3. "Lack of communication. Nothing in common."
4. "He scares me."
5. "Won't listen to my side of the story."
6. "Too quick to criticize."

There were 39 questionnaires that would like to change some of our habits, and these are what they would change:

1. "Being too much of a perfectionist."
2. "Lack of understanding."
3. "Too rushed to consider all aspects of a situation."
4. "Give up smoking."
5. "Give up card playing."
6. "Give up swearing when things go wrong."
7. "Give up trying to stop smoking and trying to lose weight. Makes him too irritable."
8. "Too many spur of the moment chores. If he works, everyone else has to."
9. "Too quick to criticize." (Frequently mentioned.)
10. "Should be more open minded."
11. "Take better care of himself."
12. "Not eat so fast."
13. "Too irritable and moody."
14. "Disdainful attitude towards sports and pop music."
15. "Be more punctual."
16. "Stop ignoring you when you try to talk to him."
17. "Be more communicative."
18. "Not so dictatorial." (Several listings.)
19. "Curb some outside non-medical activities."
20. "Better bill collecting habits."
21. "Stop medical talk at mealtimes."
22. "Overemphasis on school grades."
23. "More willing to listen to my side of the story."
24. "Take more interest in my activities."
25. "Should relax more."
26. "Should develop interests outside of medical things."

The last question asking for a summary of opinion of life in a physician's family and of doctors as parents produced food for thought. The great majority said they liked the life, thought it was interesting, helpful, educational, and would not want to swap it. The opinions as to doctor fathers varied from a "complete failure" to "if he were any better, he would have to be God." Most of the questionnaires indicated that they thought we did a pretty good job.

Again, one was impressed with the strong feeling of pride in father's ideals and dedication to helping others, the self-sacrifice involved, the long hours of hard work, the responsibilities toward patients which so many of our children credit us with. They regretted the lack of time to spend with father, but they did not resent it. "Skip the ball games. The vacations aren't important. Dinners are just food. But hold for your son the highest, most noble image there is, that of a Doctor of Medicine." This was from a boy in the 15 to 20 years.

Many expressed the thought that being physicians had nothing to do with the kind of fathers we were, that a man would be the same kind of father no matter what his occupation.

There was a continual plea for more communication and more understanding, but this too seemed to be unrelated to being a physician. The majority said their fathers were excellent parents, and many spoke of the importance of their mothers in helping dad to be a good father.

As one put it, "Dad does O. K. as a parent most of the time, and mom's not bad either." Another says, "A physician's greatest asset is an extremely good wife." Still another offers this observation, "For his devotedness to his profession I early developed a great respect for my father, and any child who can do this at an early age can face and accept the minor trauma of a thwarted camping trip or day at the lake. In my opinion, it is the mother's responsibility to instill this respect by conscientiously explaining the situation and by showing adaptability herself to irregular hours and broken plans." This statement seemed quite to the point, "It is a very necessary thing that a doctor's child must have an enduring understanding that his father's profession, by its nature, makes more demands, interferes more with family activities, than other professions, and must be primary in cases of conflicting interests."

During these summations, many restated the disadvantages of being a physician's child, recalled the advantages and decided they more than outweighed the complaints.

Conclusions

It was a relief to find that no great problems seem to exist in the critical areas of time with family, interrupted plans, and participation in outside activities. Most of the children thought that they did not have many plans upset, that their fathers did have enough time to spend with the family, and those who thought otherwise did not resent it. Most of the questionnaires said that they thought their fathers did miss more school, social, and church activities than did non-physician fathers, but not many were bothered by it. Since these aspects of our lives seem relatively unchangable, it is fortunate that there seems no need for change as far as our children are concerned.

In general, most of the complaints and disadvantages mentioned were those that any group of children might come up with regardless of father's occupation. The lack of interest, lack of understanding, excess of criticism, are all common to any study of parent-child relationship. So an attempt was made to isolate those complaints that might be directly related to our being doctors. Some of these we should be able to correct or alleviate.

The telephone seems to bother our children as much as it does us. Certainly a phone for the family should be provided, if possible, and it does seem unreasonable to expect our families to answer the office phone after hours. The expense of an answering service or a recording device, when we are not available, might be cheap enough for the relief it would give to the rest of the family.

Having an office in the home has always seemed like a good idea to some of us. One can be more available to the family, skipping into the household between patients for a quick visit, etc., but now it seems this is wrong. At least our children think so. If circumstances demand an office in the home, then we should try to have separate entrances, phones, baths, good sound-proofing, with as little contact between office and family as possible.

No medical conversation at mealtimes. This is easily corrected, and should improve appetites and dispositions.

Becoming irritable and disagreeable when we are on call or on service at the hospital, or when we have those days when everyone is sick and nobody is getting better, seems to be a pretty common complaint. "Taking it out" on our children after a busy and trying day is a problem peculiar to pediatricians but could be a release mechanism for any doctor. A little restraint along these lines would be welcome by our families.

Taking more interest in our children when they are ill brings up the old problem of caring for ones family. Most doctors don't like to do this, and rightly so, but perhaps a little more show of concern would not be out of order. Our children have indicated that they expect it, and when we don't provide it, they think it means indifference or lack of confidence in our own ability. Perhaps we should explain to them in more detail why doctors should not try to take care of their own family illnesses in most instances. I'm sure it is a mistake to be indifferent, and equally sure it is a mistake to be overly concerned. One of my own mistakes is to get angry about it! I don't know why, but I tend to get furious inside when one of my own becomes ill. I think I have noted this in other physician fathers, too! Whether it is due to worry or concern or fear is hard to tell, but we tend to jaw and sputter about it, and I am sure this rubs off on the sick one. There must be a happy medium here somewhere!

The question of being too dictatorial is interesting. One wonders if this might be a carry over from ordering nurses, aides, and technicians around and expecting them to carry out every order promptly and efficiently. It seems reasonable that after years of this on wards, in offices, and in laboratories, it might start to creep into our family relations. It should be easy to correct on our part.

Quite a few of the questionnaires expressed a wish to know more about what a physician does, what his work encompasses. This should be a painless thing to do. Maybe an explanation of our schedules, problems, work, frustrations, triumphs, and tasks be they mundane or exotic, might help our families to understand better our absences, lack of punctuality, or our dispositions.

It should be easy to provide some area in our homes where the friends of our children would feel welcome, and not "too scared to come."

Developing interests outside of the medical world should be a duty for every physician, for his own sake as well as his families relief. It is difficult to do, but it seems as if everyone would benefit from it.

It seems we could all establish better lines of communication with our families. This is a frequently mentioned complaint against us. We either fail in not providing it when we have time, or not taking the time when we can. Several children said that their fathers had set up a half to one hour period every week for a question and answer period for them alone. This they could count on, and they didn't mind that dad was tied up or unavailable the rest of the week. They stated that this hour had remained a highlight in their memories. This sounds like a splendid solution as long as one does not let the hour become a disciplinary time. We may not all be Longfellows, but we should all be able to supply a "Children's Hour."

The last observation is a pertinent one. So many of the questionnaires said they would like their fathers to take better care of themselves physically. The solution is simple and probably ignored by most of us. Many children said that although extra cars, fancy clothes, summer camps, big allowances were nice to have, they were not as important as having dad around for a good long time, and, please would he remember it.

I would like to close with a quote from Kahlil Gibran's book *The Prophet*:

"And a woman who held a babe against
her bosom said, 'Speak to us of children.'
And he said:
Your children are not your children,
They are the sons and daughters of
Life's longing for itself.
They come through you but not from you,
And though they are with you yet they
Belong not to you.

You may give them your love but not
Your thoughts,
For they have their own thoughts.
You may house their bodies but not
Their souls,
For their souls dwell in the house of
Tomorrow, which you cannot visit, not
Even in your dreams.
You may strive to be like them, but seek
Not to make them like you.
For life goes not backwards nor tarries
With yesterday."

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Pancreatitis and Acute Rheumatic Fever

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The cause of abdominal pain in acute rheumatic fever is unknown except in those instances with congestive heart failure.[1] The simultaneous occurrence of acute pancreatitis and acute rheumatic fever in the patient to be described raises the question as to whether this is simply a chance relationship.

CASE REPORT

A 15-year-old male was hospitalized 3/14/62 with a 2½ week history of mid-back (low interscapular) pain, five days of abdominal pain, nausea and vomiting. At the time of admission, no localizing abdominal findings other than slight tenderness of the rectus muscle insertions at the pubis were detected. A urine diastase was strongly positive (1:512, normal up to 1:32). Upper G.I. series on 3/14/62 showed irritability and spasm of the third portion of the duodenum compatible with pancreatitis. The patient continued to complain of nausea, vomited and retched over the next 10 days. Repeat urinary diastase on 3/22/62 was still slightly elevated, 1:64. No serum amylase was done. Bilirubin on admission and repeated one week later was less than 1 mg%. Repeat upper G.I. series done 2½ months later was normal, as were gallbladder X-rays. Intravenous urogram was also normal.

Complaints of pains in the legs were mentioned prior to admission but no objective findings were present in the joints until one week after admission. This was during the period of acute pancreatitis described above. Painful swelling developed in the wrists, both knees, shoulders, right elbow and joints of the right hand. There was redness and exquisite tenderness associated with these findings. A joint remained painfully swollen and hot from 2 to 4 days. All joint symptoms and signs cleared within 1 week of salicylate therapy, which was begun on 3/24/62. There has been no recurrence of joint symptoms since stopping salicylates 6 weeks later. The uric acid was 4.4 mg%, rheumatoid arthritis serum test (latex fixation) was weakly reactive, the anti-streptolysin titre was 2500 units. A throat culture two weeks before admission showed normal flora, two blood cultures and repeat throat culture during the early phase of illness were negative. Additional evidence of acute rheumatic fever was the development of a louder systolic murmur than had been present before admission, first noted to be holosystolic and heard at the base as well as the apex on 4/1/62, then the development of a diastolic decrescendo murmur along the left sternal border, first heard on 4/16/62. These findings were corroborated by a cardiologist. There was associated widening of the pulse pressure, initial blood pressures were 130/80; later pressures were in the region of 150/70. Serial electrocardiographs showed slight prolongation of the QT (0.42 seconds with a rate of 65). Heart size was normal by X-ray.

SUMMARY

This patient with acute rheumatic fever presented with an acute abdomen. A diagnosis of acute pancreatitis was made and one week later a migratory polyarthritis developed and subsequently the diagnosis of acute rheumatic fever established. This case is reported in the hope that others confronted with patients experiencing abdominal pain in association with rheumatic fever will study these patients for evidence of pancreatitis.

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Return of Viability of the Intestine With Restoration of the Blood Supply*

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INTRODUCTION

The surgical attack on vascular disease in all areas of the body has become more aggressive during the past decade. The prognosis of any disease depends on the stage at which it is treated. The prognosis of vascular occlusion is likewise determined by the extent and duration of the local lesion. The ultimate outcome is also affected by the location of the local lesion and the condition of the remaining portions of the arterial system. During the past five years attention has been focused on the superior mesenteric artery.

On August 25, 1951, the first successful embolectomy of the superior mesenteric artery was performed.[1] The case was not reported in the literature until 1958. In 1959 the first case of intestinal angina was successfully treated surgically.[2] In this particular case the intestinal blood supply was deficient, but a complete occlusion was not present. Although segments of the intestine were dusky, black infarction was absent. Prior to five years ago the surgical procedure for intestinal infarction was resection. With increased interest in this subject, animal laboratory work, and operating experience, many articles have appeared in the surgical literature changing the surgical treatment. The first operative maneuver is to determine whether the intestinal occlusion is arterial or venous. This is easily ascertained by the presence or absence of pulsation in the mesenteric arcades. Arterial occlusion is the most common. Venous occlusion usually only exists in the presence of infection or mechanical obstruction by adhesion bands and incarcerated hernias. If arterial pulses are absent, the second step is to relieve the arterial occlusion. This is accomplished by exposure of the superior mesenteric artery and identifying the level of the block of the arterial blood flow. Only after the occlusion has been treated, is the status of the intestine evaluated and a decision whether or not resection is necessary made. This can be a very difficult decision. If there is any question, resection is not done at the initial procedure, but a second look procedure is performed in 24 to 48 hours. It is unbelievable until one sees how a black intestine can return to viability. The author+ had this experience recently. Review of the literature did not reveal a case of complete occlusion with black intestine at the time of surgery with return of viability of the intestine after restoration of blood supply. Therefore it seems worthwhile reporting the details of this case.

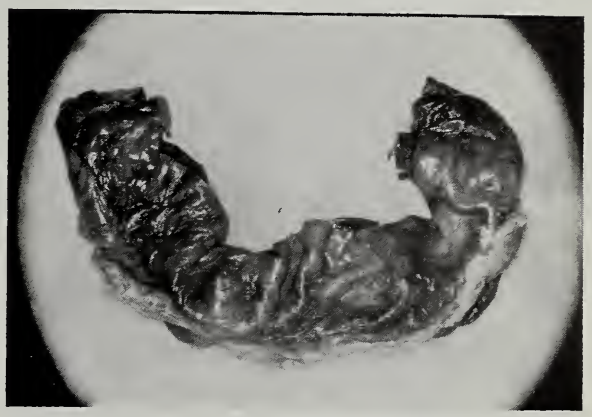


FIG. 1. Serosal surface of jejunum showing infarction. No restoration of blood supply and viability.



FIG. 2. Mucosal surface of jejunum showing infarction.

CASE PRESENTATION

An 84-year-old female was seen in surgical consultation in the evening of the day of admission to the hospital because of abdominal pain, beginning distention of the abdomen, and the presence of nausea and vomiting. Although patient appeared acutely ill, she denied having very much pain in the abdomen. There was a history of congestive heart failure during the past year with apparent good response to medication. She had felt fairly well up until three weeks prior to admission when she noted weakness and a decrease in her appetite. The first onset of abdominal pain was said to be one day prior to admission when she had the sudden onset of very severe pain causing her to pass out. On the day of admission to the hospital, patient had taken very little liquids by mouth because of her nausea and passed only about 2 to 3 ounces of urine.

Patient was a very seriously ill, elderly woman, well preserved for her 84 years. The mucous membranes of her mouth were slightly dry, the skin had good turgor with no evidence of loss of subcutaneous tissue or presence of dehydration. The heart rhythm was noted to be irregular and the pulse rate was

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FIG. 3. Serosal surface of ileum showing return of viability.

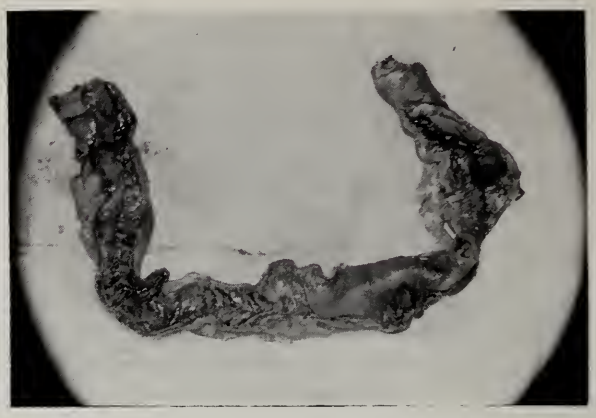


FIG. 4. Mucosal surface of ileum showing return of viability.

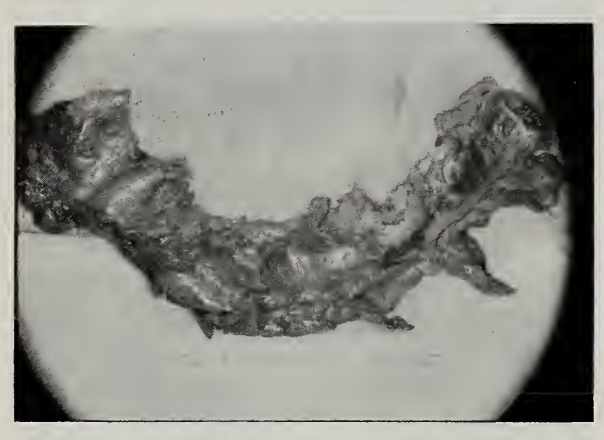


FIG. 5. Serosal surface of ascending colon showing return of viability.



FIG. 6. Mucosal surface of ascending colon showing viable areas.

around 100. The blood pressure on admission early in the day was 170/90 and at the time of being examined was 95/60. The radial pulse was of poor volume. The abdomen was found to be softly distended with marked tenderness over the lower half of the abdomen. Peristalsis was absent; no bruit could be heard. No hernias could be found in the umbilical, femoral, or inguinal regions. Liver and spleen could not be felt and there was no CVA tenderness. There was no mass palpable in the abdomen. A Cantor tube was inserted for deflation of the bowel and 900 cc. of greenish material obtained with some softening of the abdomen during the next two hours. Patient would not leave the tube in place, requiring its reinsertion on two occasions and finally a Levine tube was inserted. With the decrease in distention, the abdomen could be more easily evaluated and it was now determined there was definitely more tenderness in the left lower abdomen. Rebound tenderness was not present. She did have three scars on her abdomen of previous operations, including a midline hysterectomy scar, a right lower quadrant appendectomy scar, and an upper right abdominal scar of previous cholecystectomy. All of these scars were well healed without evidence of hernia. Rectal examination revealed no intraluminal mass. The stool was normal color and negative for occult blood. Sigmoidoscopy was carried out to 20 cm. in an unprepared colon; no lesion could be seen. It was noted that the popliteal arterial pulsations were present bilateral, but no pedal pulses could be felt.

Review of the admission x-rays: The chest showed clear lung fields with enlargement of the left ventricle. The abdomen showed small bowel distention consistent with that of obstruction. The admission laboratory work revealed an elevated white count with shift to the left and an elevated hematocrit indicating the status of dehydration. The total white blood count was 23,900 and the differential was polys 61, band forms 28, lymphs 10, monocytes 1. The hematocrit was 46%. A urine was unobtainable.

The surgical opinion at the initial examination and evaluation the following four hours was that this elderly lady was a poor surgical risk, and possibly could be improved by intravenous fluids, bowel suction, antibiotics and vasopressor. The initial impression was: 1. Probable diverticulitis of the sigmoid with perforation and peritonitis. 2. Possible mesenteric artery occlusion.

Twelve hours after the initial examination this patient was obviously showing no improvement. She had received 3000 cc. of fluids with electrolytes intravenously. The blood pressure was 80/50 with a feeble pulse of 110. The abdomen had been deflated by suction. Examination revealed more tenderness in the left and mid lower abdomen with rebound tenderness now being demonstrable. Barium enema had been scheduled, but was cancelled and preparation for surgery carried out. It seemed quite logical that this patient either had mechanical obstruction to the intestine requiring surgical intervention for a gangrenous bowel or mesenteric artery occlusion. If it was mechanical obstruction, survival might be possible; if the latter condition existed, survival was not expected. Neo-Synephrine® drip was started and the systolic blood pressure was brought up to 120.

The entire surgery was carried out under local anesthesia with sedation. At operation, the jejunum, ileum, ascending colon, and right half of the transverse colon were black and lifeless. The peritoneal cavity contained a large amount of serosanguinous fluid. Both the operating surgeon and the assistant felt that any procedure was futile and death inevitable. Examination revealed no pulsation of the superior mesenteric artery. Transverse mesocolon was opened on its underside, exposing the

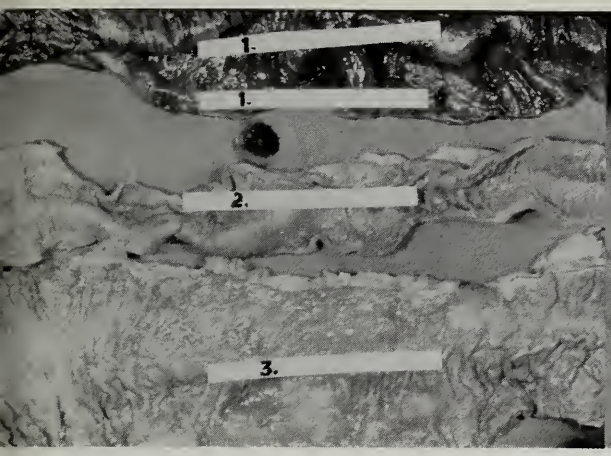


FIG. 7



FIG. 8

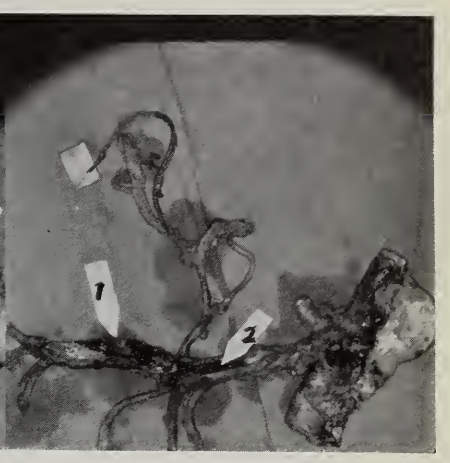


FIG. 9

FIG. 7. Mucosal surfaces of [1]jejunum, [2]ileum, and [3]ascending colon to show the darkness of the jejunum indicating its arterial deficiency.

FIG. 8. Superior mesenteric artery with attached segment of aorta.
mc–middle colic artery
j–jejunal arteries
Tag 1 points to Teflon graft covering arteriotomy site.
Tag 2 points to adherent clot on the wall of the superior mesenteric artery extending into and completely occluding the jejunal branches.

FIG. 9. Arrow 1 pointing to the site of the Teflon graft which has been removed to show the patent lumen of the superior mesenteric artery.
Arrow 2 showing occlusion of the jejunal arteries.

superior mesenteric artery, duodenum and associated structures. The site of the vascular occlusion was isolated and a clot removed with good flow of blood from the aorta. The arteriotomy site was closed with a Teflon patch. While carrying out this procedure, the proximal and distal portions of the superior mesenteric artery were clamped. Initially, release of the proximal clamp did not result immediately in a good flow of blood. The patch was lifted and this explored further by suctioning and endarterectomy. Because of patient's grave condition, thorough exploration of the proximal segment of the superior mesenteric artery was not carried out. However, at the completion of the suturing of the Teflon patch and release of the clamps, a satisfactory blood flow was obtained through the graft and into the distal portion of the superior mesenteric artery. Further observation of the color of the intestine over an adequate period of time for recovery of its circulatory state was not done for reasons previously stated. It was the opinion of the operator that some improvement in the color of the intestine did take place in this short interval of time.

It seemed that this patient would be dead within 24 hours. The subsequent course was quite amazing. In spite of the diseased bowel known to be present and the effect on renal function demonstrated in the past by many investigators, this patient continued to have a fair postoperative output of urine. For 12 hours prior to surgery it was 500 cc. The day of surgery it was only 40 cc., but patient did not receive 1500 cc. of her ordered intravenous fluids, because the cutdown was disrupted. The following 24 hours, being the first day postoperatively, patient had an output of 320 cc. for an intake of 2000 cc. The second postoperative day she had an output of 1400 cc. with an intake of 4000 cc. From this I think we can assume her kidney function remained fairly good. Twenty-four hours after surgery the Neo-Synephrine drip was discontinued and patient's blood pressure maintained itself between 120 and 140 and finally stabilized around 140 systolic. Her progress was satisfactory through the third day following her surgery. She had passed gas rectally and had audible peristalsis since the second day. In the evening of the third day she died suddenly. One hour prior to her death her blood pressure was 140/90. She appeared well and had talked with her relatives. Death was so sudden the nurse in attendance did not have time to check her blood pressure or administer oxygen.

POSTMORTEM FINDINGS

I. Gross and microscopic examination of intestine.

II. Gross and microscopic report on the superior mesenteric artery.

The postmortem examination was performed three hours after death, and the peritoneal cavity contained about three liters of reddish fluid. Some of the peritoneal surfaces were lack-luster but no fibrin accumulation was noted. The duodenum seemed somewhat dusky in color and somewhat distended. The jejunum was distended to a circumference of seven centimeters, and the serosal surface black and lack-luster. When the jejunum was opened, the mucosal surface was also black. Microscopic study confirmed the appearance suggested by FIGS. 1 and 2, that of infarction, with preservation of the outline of the muscle wall and mucosa, but with loss of the usual staining reactions, loss of nuclear detail, and with an intense acute inflammatory reaction.

The ileum showed a gradual loss of the black lack-luster appearance of the jejunum so that the distal half showed a nearly normal color of the surface and some return of luster. The mucosal surfaces were also normal, and there was no blood in the lumen. FIGS. 3 and 4 show the serosal and mucosal surfaces of the ileum.

The fairly normal appearance of the bowel persisted into the ascending, transverse, and descending colon and again the mucosa appeared normal. FIGS. 5 and 6 demonstrate this apparent viability, and microscopic study revealed a generally well-preserved bowel with occasional focal areas of mucosal necrosis. FIG. 7 gives a comparative study of the gross appearances of the jejunum, ileum, and ascending colon.

The aorta was studied and showed moderate calcific athero-



FIG. 10. A close-up of the Teflon patch on the arteriotomy opening.

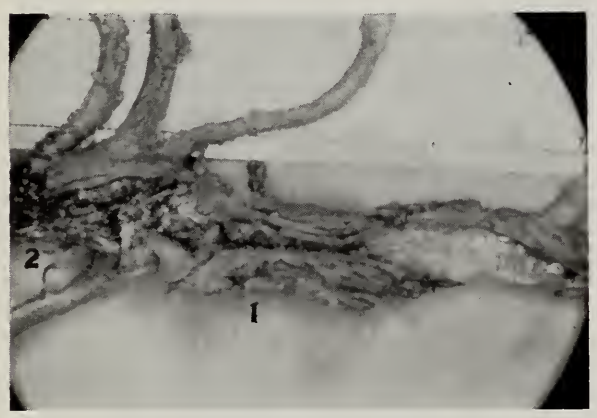


FIG. 11. 1. Area of arteriotomy and patch.
2. Old thrombus adherent to and occluding the jejunal branches.

sclerosis with several eggshell type calcific plaques. The orifices of the major vessels were not encroached upon by the process. The superior mesenteric vessel, particularly, showed no narrowing of the orifice. Beginning at a point eight centimeters from this orifice the surgically created wound in the wall of the artery was noted and the Teflon patch was seen to be in place. Two and one-half centimeters proximal and for a distance beyond this wound, adherent antemortem clot partially occluded the lumen and tended to extend into and completely occlude the jejunal branches. The sections confirmed the antemortem nature of the clot and showed atherosclerosis of a moderate degree as the background lesion for the thrombosis. FIGS. 8 through 12 demonstrate the superior mesenteric artery segment under discussion, and the relationship of the clot and the arteriotomy to the various branches. Microscopic sections of the mesentery were unremarkable and in particular showed no evidence of small vessel intimal hyperplasia, or thrombotic occlusion.

There was no pulmonary embolism. The heart was essentially normal, and thus no explanation for the sudden death was found.

SUMMARY OF CASE REPORT

From the history, surgery, and postmortem findings, one can postulate the following sequence of events: 1. Thrombosis of superior mesenteric artery extending into the jejunal arteries with partial occlusion of the superior mesenteric artery and total occlusion of the jejunal arterial branches present for a questionable number of



FIG. 12. Close-up of adherent clot in the superior mesenteric artery extending into the jejunal branches 1, 2, and 3. Arrow marks the clot. 4. Middle colic is patent.

months. 2. The jejunum remained viable because of collateral blood supply from duodenum and adjoining ileum arteries. 3. Fresh thrombus or embolus on top of the old thrombus completely occluding the superior mesenteric artery producing intestinal infarction and peritonitis. Surgical findings of black jejunum, ileum, cecum, ascending colon and right half of the transverse colon. 4. Superior mesenteric arteriotomy and removal of fresh thrombus bringing about a return of blood flow to the ileum and colon. 5. The jejunum failed to obtain sufficient blood flow, due to the total occlusion of the jejunal branches by the old adherent thrombus, for a return to viability. 6. Patient's postoperative course was quite satisfactory considering the moribund state preoperatively. The sudden death is still not definitely explained, but postulated to be due to cardiac failure from either standstill or flutter. It is quite possible a second procedure could have been done and tolerated within 48 hours to allow for removal of the dead jejunum.

DISCUSSION OF SUPERIOR MESENTERIC ARTERY OCCLUSION

In the development of this discussion, four principles will be stressed. They are as follows: 1. Surgical treatment must be in the early occlusive phase. In the past surgical treatment of superior mesenteric artery occlusion has been too late, only after complete occlusion with small and large intestine infarction and peritonitis resulting in 100% mortality. 2. The surgical attack should be on the pulseless superior mesenteric artery. The cause, if arterial occlusion, should be remedied rather than treating the resulting pathology, bowel infarction. Bowel viability may be difficult to determine at the initial procedure and intestinal resection may be contraindicated. 3. Because of the difficulty of determining viability or capacity for return of viability, second-look procedures have been advocated.[3] 4. The diagnosis of mesenteric artery occlusion is by a process of elimination.

The surgical lesion includes the majority of the patients. There is a rare type of endartery occlusion which

will be discussed. Superior mesenteric artery occlusion may be gradual or sudden. It has been demonstrated in the human and animal subjects that the intestine can survive gradual occlusion of the celiac and superior mesenteric arteries.[4] Sudden occlusion, however, results in bowel necrosis. To make matters worse, sudden occlusion is usually a cardiac patient who has either had a recent myocardial infarction, is fibrillating, or has been in recent failure. There have been no reported cases in the literature of salvage of patients after total intestinal infarction of the superior mesenteric artery supply as in the clinical case presented. The only sudden occlusion cases that have been salvaged are usually those already in the hospital who are operated on early when the intestine may show early changes of circulatory embarrassment but not necrosis.

An example of the gradual occlusion would be the intestinal angina patient. This may be due to a plaque at the superior mesenteric artery origin or elsewhere in the lumen. Progressive occlusion may then develop with thrombosis formation on the plaque. This is the stage that should receive our surgical treatment. The clinical picture is an elderly patient complaining of postprandial distress. This consists of crampy abdominal pain coming on fifteen to thirty minutes after eating. The severity of the pain is directly proportional to the amount of food ingested and lasts from one to three hours. As a result of the pain, the patient eats less and loses weight. Abdominal examination, laboratory studies and x-rays of the gastrointestinal tract are not remarkable. A systolic bruit may be heard in the upper abdomen. Tenderness does not develop until infarction of the intestine is present. Even though the branches to one segment of the small intestine are completely occluded as in the case presented, there is adequate collateral anastomosis of the intestinal branches to maintain intestine viability. This was visualized by Reiner, Rodriguez, Platt, and Schlesinger in 1959 who reported on the injection of the mesenteric arteries.[5] In addition to the arterial arcades of the duodenum, jejunum, and ileum in the mesentery there is communication between the celiac and superior mesenteric arteries through the superior and inferior pancreaticoduodenal arteries. Also there is a major union in the majority of people through the marginal artery of Drummond which unites the superior and inferior mesenteric arteries.

Another anatomical lesion producing the intestinal anginal syndrome and presumably leading to infarction of the intestine is intimal hyperplasia of the small mesenteric arteries. This lesion has been recently recognized[6] and is admirably demonstrated when it is present by a section of mesentery taken one centimeter from the attachment to the bowel and parallel to the longitudinal aspect of the bowel. This lesion, which is of course not subject to therapeutic management, is to be suspected when exploration fails to demonstrate arterial occlusion.

The intestinal angina syndrome is similar to angina pectoris and claudication of leg muscles, except that in this instance, intestinal muscle is receiving insufficient blood during working hours and relating the fact to the patient by the symptom of pain in the abdomen. The information gained from autopsy and surgery has encouraged surgical procedures on the superior mesenteric artery.[7] Frequently there is segmental occlusion in the proximal portion of the superior mesenteric artery with absence of disease in its distal portion and smaller branches rendering itself to instrumentation and removal of the occluding clot.

The clinical diagnosis is usually by a process of elimination of other diseases. Naturally one thinks of this problem with a typical story as previously presented. The diagnosis can be made preoperatively by aortogram with exposures in the lateral position.[8] The aorta will obscure the celiac and superior mesenteric arteries with an anterior-posterior exposure because of their anterior position. Mikkelsen and Berne of the University of Southern California School of Medicine prefer to omit aortography in favor of direct surgical exploration. Their reasons are: 1. Time, expense, and potential hazard of the aortogram. 2. The urgent obligation for abdominal exploration in a patient with unexplained progressive abdominal pain.

The surgical treatment has included the same techniques used in arteries elsewhere, that is: embolectomy, endarterectomy, by-pass graft, and a combination of these procedures. The surgical exposure is obtained through the inferior surface of the transverse mesocolon or through the gastrocolic ligament.

Review of Mesenteric Thrombosis and Infarction Cases at the Eastern Maine General Hospital from January 1959 through November of 1963. (See Table 1)

There were twelve cases of superior mesenteric artery occlusion during this interval of almost five years of a total of 60,000 hospital admissions. This would be one case of mesenteric artery occlusion in 5,000 admissions. This is certainly not a frequent condition. The age group varied between 58 and 91 years; the majority being over 75 years of age. The sex distribution was about even. All of the cases died. Nine of the cases were proven to have a superior mesenteric artery occlusion and intestinal infarction by either surgery or postmortem examination, or both. In all cases an intestinal resection was impossible because of involvement of the entire small intestine and right half of the colon. The patients all had severe abdominal pain, nausea, and vomiting. Five of the twelve cases were in moderate to severe shock, described by the examiner as moribund. Of the seven postmortem examinations the occlusion of the superior mesenteric artery was described as at the origin in four, in the fifth case it was 2½ centimeters from the origin, and in the case discussed today it was approximately 6 centimeters from the origin. Only three of the twelve cases were operated upon and in only one case was an attempt made to restore circulation. Seven of the twelve cases had

TABLE 1

Admission Date	*Age*	*Sex*	*Clinical Features*	*Preop Dx.*	*Op. Findings Surgery*	*Results*
4/15/59	69	M	Vomiting, abdominal pain one day prior to admission, fibrillation, BP 122/74, Pulse 150.		No Surgery	Died 4th day after admission. Postmortem: Obstruction of superior mesenteric artery 2½ cm. from origin by a clot, probable embolus. Thrombus in left atrium.
1/30/60	76	F	Sudden onset of abdominal pain, nausea, vomiting two hours postprandial, 24 hrs. prior to admission. BP 108/80, Pulse 130, fibrillating, WBC increased with a shift to the left.		No Surgery	Died 24 hrs. after admission. No postmortem.
5/60	88	F	Abdominal pain 3 days prior to admission. Four to five months decrease of appetite and weight loss. BP 120/74, Pulse 116, Heart rhythm regular. Pedal pulses not present.		No Surgery	Died 20 hrs. after admission. Postmortem: Occlusion of superior mesenteric artery at its origin, small intestinal infarction.
8/6/60	84	F	Moribund, abdominal pain with radiation to the back. Diffuse tenderness of the abdomen. Two week history of abdominal pain, peristalsis decreased, heart rhythm irregular. Clinical Dx: Diverticulitis with perforation. Absent popliteal and pedal pulses bilateral. X-rays showed gas-filled small intestine.		No Surgery	Died 12 hrs. after admission. Postmortem: Small intestinal infarction.
10/28/60	69	F	Admitted with hemaplegia due to CVA, comatose, diabetes mellitus.		No Surgery	Died 7 days after admission. Postmortem: Generalized arteriosclerosis, myocardial infarction, superior mesenteric artery occlusion at the origin, small intestine and right colon impending gangrene.
4/1/61	91	F	Admitted with intestinal obstruction abdominal distention.		No Surgery	Died two hrs. after adm. Postmortem: Occlusion of superior mesenteric artery at the origin, infarction of small intestine.
4/19/62	58	M	BP 100/60, Pulse 110, considered poor risk for surgery. Abdominal pain, distention, two days prior to admission. Absent peristalsis, generalized tenderness. No pedal pulses, probable mesenteric artery occlusion. X-rays show small and large bowel distention.		No Surgery	Died 28 hrs. after hosp. adm. No postmortem.
6/28/62	76	M	Crampy abdominal pain and vomiting, 2 days prior to admission.	Small bowel obstruction	Surgery one day after adm. Small and large intestine infarction found, and abdomen closed. No superior mesenteric artery surgery.	Died 5½ days postop. No postmortem.
7/2/62	74	M	Abdominal pain and vomiting 24 hrs. prior to admission, abdominal distention, no peristalsis. X-ray: Ileus.		No surgery, pt. moribund on adm.	Died 1½ hrs. after admission, no postmortem.
11/19/62	80	M	Abdominal pain, nausea and vomiting, 30 hrs. prior to admission, BP 180/100, Pulse 120, fibrillation, generalized tenderness of abdomen. Blood in rectum. WBC increased with shift to the left.	Peritonitis	Gangrene of small intestine, right colon. Superior mesenteric artery pulseless, no attempt made to clear artery.	Died 8 hrs. after adm. and immediately postop. No postmortem.
3/4/63	88	F	Cholecystostomy for acute cholecystitis. 1st day postop, irregular cardiac rhythm. 9th day postop abdominal tenderness, 12th day postop distention of abdomen. 13th day postop patient died.		Cholecystostomy for acute cholecystitis and cholelithiasis.	Died. Postmortem examination: obstruction at origin of superior mesenteric artery by plaque. Small and large bowel infarction.
11/14/63	84	F	Case presented.			

definite cardiac pathology and six were fibrillating. One of the cases was admitted as a hemiplegic due to a cerebral vascular accident.

Conclusions

1. Report of a case of complete occlusion of the superior mesenteric artery with infarction of the jejunum, ileum, ascending colon, and right half of the transverse colon.

2. Black, non-viable intestine at surgery returning to viability after removal of occluding clot.

3. The jejunum did not return to a healthy state as the three jejunal arteries were occluded by an adherent, long-standing thrombus.

4. An elderly woman in shock preoperatively who contrary to expectation survived surgery and had a benign postoperative course for three days.

5. Restoration of superior mesenteric artery blood flow should be done before total occlusion and intestinal infarction.

6. Early stages of superior mesenteric artery occlusion are difficult to diagnose. In order to have earlier surgery and increase our salvage rate, we must think of superior mesenteric artery occlusion in every patient over 60 years of age complaining of abdominal pain.

7. Surgery should be directed to relieving the superior mesenteric artery occlusion.

8. The frequency of superior mesenteric artery occlusion will increase as the longevity of the population increases.

9. Old age and other systemic disease, cardiac, renal, and pulmonary are complicating factors usually present with superior mesenteric artery occlusion, but should not deter one from active attempts at prolonging life.

10. The majority of patients may still be operated on late, after total occlusion and infarction. Attempt should be made to restore blood flow in the superior mesenteric artery as there may be a possibility in the future of salvaging one of these otherwise fatal cases.

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* * * *

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Aortic Stenosis and Gastrointestinal Hemorrhage

Report of Four Cases

Mason Trowbridge, Jr., M.D.*

Either aortic stenosis or gastrointestinal hemorrhage can be a complex clinical problem. Occurring together they really tax the clinical acumen of the internist, surgeon, and radiologist. The patient's precarious cardiac status may render surgery or even radiographic studies hazardous. Transfusions are necessary, and one is caught between the Scylla and Charybdis of congestive heart failure and hypovolemia.

Case 1

An 80-year-old white woman complained of weakness and dizziness. She had been known to have heart disease for 12 years, and 7 years previously the diagnosis of aortic stenosis had been made. At that time she complained of anginal pain and syncope. Two years previously she had "passed out for an hour". In spite of digitalis, diuretics, and salt restriction she developed "asthma" a few months prior to admission. Because of the history, hemoglobin 9.0 Gm./100 ml., and a guaiac positive stool she was admitted for study.

Physical examination showed her to be pale, dyspneic, and apprehensive. Blood pressure: 130/80. Pulse regular at 100. Positive physical findings included slight venous engorgement of the neck, minimal changes of the optic fundus, the apex of the heart in the anterior axillary line, and a grade IV diffuse systolic ejection murmur loudest in the aortic area. A2 was not heard. Hematocrit 26%. WBC: 7,750 c. mm^3 with a normal differential. Smear showed hypochromia, microcytosis, anisocytosis, and poikilocytosis. Neither in this case nor the other cases was a disturbance of the clotting mechanism demonstrated. X-RAY: Enlargement of left ventricle, calcification of the mitral and aortic valves. IMPRESSION: Aortic stenosis. Lung fields clear. G.I. series and barium enema (repeated) revealed moderate duodenal scar formation and a 15 mm. protrusion of the terminal ileum into the caecum. The radiologist did not consider this of clinical significance. Sigmoidoscopy negative to 15 cm. COURSE: Patient was given 3 transfusions of 500 c.c. without cardiac decompensation. The hematocrit was 35% and the stool guaiac 2+ at time of discharge.

Case 2

A 95-year-old white spinster, who had been surprisingly active in spite of advanced rheumatoid arthritis, developed melena which persisted for 10 days. Her gastrointestinal history was otherwise negative. Previously atrial fibrillation had been controlled by digitalis with no evidence of congestive heart failure. Her hematocrit four months prior to admission was 42%.

Physical examination revealed her to be about her stated age of 95. She was in no obvious distress. Although appearing almost exsanguinated she was haranguing the accident room personnel about President Kennedy's taking us into socialized medicine. Blood pressure: 120/40. Fibrillating 100. Heart: no enlargement. Grade II aortic systolic murmur. Chest clear. Liver down 4 cm. Hematocrit 14% with normal WBC and differential. BUN 13 mg.% and stool guaiac negative at time of discharge. X-RAY: Examination of the upper and lower gastrointestinal tract was negative except for extensive diverticulosis. Slight cardiac enlargement. ECG: atrial fibrillation. S-T and T changes suggestive of either digitalis effect or hypokalemia. COURSE: Discharged on 8th hospital day after three transfusions administered without inducing congestive heart failure.

Second Admission: Three weeks after discharge, having had positive guaiac stools, she passed bright red blood rectally. She was again transfused from a hematocrit of 22% to 36% with only the development of a few rales in her chest.

Third Admission: She had passed a large, gelatinous, reddish black, guaiac-positive stool. She was again transfused, and no further efforts to determine the source of bleeding were made.

At age 97 she died in a nursing home after developing fever, paroxysmal coughing spells, and rales at both bases. AUTOPSY: Calcific aortic stenosis, bilateral broncho-pneumonia, and chronic pyelonephritis. There was a large diverticulum of the upper jejunum but no evidence of recent bleeding.

Case 3

A 65-year-old white housewife was admitted because of passage of bright red blood rectally. Her cardiovascular and gastrointestinal history had been negative except for hypertension. A diagnosis of aortic stenosis was made on the basis of a Grade III, coarse, systolic murmur and a blood pressure of 170/90. Hematocrit 31%. ECG within normal limits. Upper G.I. series, two barium enemas, and sigmoidoscopy to 25 cm. were negative. COURSE: Six units of blood were required. She was discharged asymptomatic on the 14th hospital day.

Second Admission: She developed a diffuse arthralgia and fever unresponsive to aspirin and prednisone. Her interval gastrointestinal history was negative. She was readmitted 19 days after discharge. The above noted systolic aortic murmur was heard with decreased A2 and transmission of murmur and thrill to both carotid arteries. X-RAY revealed enlargement of the left ventricle not present one month previously. A third barium enema was negative. I.V.P.: chronic infection of both kidneys and possible cystitis. Hematocrit 28%. Stool guaiac negative. COURSE: Because of a febrile course she was successfully treated with massive doses of penicillin. No positive blood culture was obtained.

Third Admission: Eighteen months after previous admission she had several days of recurrent left ventricular failure apparently because of inadequate salt restriction and diuretics. Hematocrit 41%. Stool guaiacs 3+ and negative. Left ventricular hypertrophy noted on ECG. COURSE: Good response to standard cardiac regime.

Fourth Admission: Recurrent pulmonary edema responding well to treatment.

Fifth Admission: Four years after initial admission had 4 days of melena. Hemoglobin found to be 6.5 Gm/100 ml. in home. Blood pressure 190/90. No signs of shock or congestive heart failure. Hematocrit 19%. G.I. series, barium enema, and sigmoidoscopy to 20 cm. negative. The patient received 7 units of whole blood with no evidence of cardiac decompensation.

Case 4

A 55-year-old white male gave a history of angina and syncopal attacks for six years. He had been considered in Boston for surgical correction of calcific aortic stenosis, but instead 13 mc. of I^{131} was given. His symptoms abated only with a vigorous diuretic and digitalization program. His past history in-

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cluded an acute episode of bleeding from a duodenal ulcer 7 years previously. Two years previously a G.I. series and barium enema were negative.

Physical examination revealed a stocky individual. Blood pressure: 120/80 to 140/100, regular sinus rhythm, and a Grade III harsh ejection systolic murmur heard over precordium, loudest in second right interspace, normal A2, rales at lung bases, minimal hepatomegaly, minimal ankle edema, and Grade II arteriosclerotic retinal changes. X-RAY revealed slight enlargement of heart and fluoroscopy revealed calcification of the aortic valve. Electrocardiograms consistent with left ventricular hypertrophy.

First Admission: Two years later. Thoracic pain considered non-cardiac.

Second Admission: One year later passage of bright red blood with hematocrit of 26% corrected with 5 units of blood. G.I. series revealed old scarring of the duodenum. Barium enema and sigmoidoscopy negative to 20 cm.

Third Admission: Two months later. Abdominal cramps and passage of bright red blood. Had been on a thiazide diuretic with potassium. No problems following transfusion of four pints of blood restoring hematocrit of 34% to normal. No further x-ray studies.

Fourth Admission: One year later admitted with crushing chest pain. There was no evidence of either gastrointestinal bleeding or myocardial infarct.

Fifth Admission: Two weeks later. Massive gastrointestinal bleeding with demonstration of a duodenal ulcer by x-ray. The surgical consultant stated that both ulcer and diverticulum of the colon should be considered as possible sources of bleeding. Uneventful correction of the anemia with four units of blood.

Sixth Admission: One month later, 13 years after first gastrointestinal bleeding, admitted with a history of two bloody stools and a hematocrit of 24%. X-RAY revealed duodenitis and diverticulosis of colon. Again anemia corrected by four units of blood. Bleeding considered to be from duodenum.

One month after discharge he died quietly in his sleep at home.

Discussion

A high incidence of gastrointestinal bleeding with aortic stenosis has been suspected in a number of quarters but has received scant attention in the literature. Williams[1] reports 1443 consecutive causes of gastrointestinal bleeding in which a demonstrable cause for bleeding was found in 93.5% of the cases. There was not a significantly higher incidence of aortic stenosis in this group than in a series of matched controls. However, of the 6.5% in whom no cause for bleeding could be found, 25% had aortic stenosis as against 5% in the matched controls. He states, "It would seem fair to speculate that aortic valve lesions have something to do with unusual forms of bleeding from the gastrointestinal tract. What the relationship may be, if any, is not yet clear and requires further study."

Goodale[2] suggests that perhaps any low output failure is likely to develop bleeding from the upper gastrointestinal tract through the mechanism described by Shipp et al.[3] This group noted abnormally high uropepsin levels following myocardial infarction with peptic ulceration. Although attention in the literature on heart disease and gastrointestinal bleeding is focused on the upper gastrointestinal tract, three of our cases reported bright red blood per rectum.

Digitalis has been suspected as a contributing cause in gastrointestinal bleeding.[4] Overdigitalization with gastrointestinal bleeding and arrythmia has been discussed by Corday et al.[5] We have seen at least three cases with congestive heart failure, arrythmias, and melena secondary to intestinal necrosis without anatomical obstruction demonstrable at autopsy. The role of the arrythmia in causing the melena was suspected only in retrospect.

Several questions were directed to Friedlich, a member of the Boston group reporting on this subject.[6] He states, "I would certainly not be any less vigorous in searching for the source (of bleeding) in the presence of aortic stenosis. In fact if there were any indication of this and a patient were coming to surgery, I would want to search very vigorously since gastrointestinal tract bleeding in the postoperative period following aortic valve replacement can be very difficult indeed. At present I have a patient who bled massively requiring suture of his ulcer but continued to bleed and another ulcer was found in the third portion of the duodenum. Most of the bleeding of which I am aware is upper gastrointestinal in origin. Certainly before doing a blind gastrectomy, I would want to exhaust every possibility including several G.I series including small bowel follow through as well as barium enemas. There have been instances, however, in which bleeding was so persistant and uncontrollable that a blind gastrectomy was done as a measure of desperation."

The central venous pressure was not used in the management of any of our cases. Our limited experience suggests that it would be of great value in restoring blood volume without precipitating congestive heart failure.

Two major clinical problems, the therapy for each possibly aggravating the other, are not easy to manage. Furthermore, the physician is subjected to vigorous pressure from the patient, his family, and other physicians. The 80-year-old woman (Case 1) had only mild anemia and cardiac decompensation when admitted. She was under the author's care because her family physician was away. In addition to the indignities such as the repeat barium enema, she developed distressing urticaria following transfusion. Word of her plight spread among her neighbors in a small town. When her family doctor returned, his voice was added to those deploring the old lady's not being allowed to go peacefully to a better world. Case 3 was even more of a problem because of the possibility of a weakened myocardium secondary to subacute bacterial endocarditis. Although a highly intelligent woman she did not even tell her family that she was having several days of melena. With some justification she did not want to go through distressing and expensive studies which with each hospital admission seemed to plumb new depths of futility. On several occasions medical and surgical consultants expressed their disapproval of extensive diagnostic procedures. Only in the case of the 97-year-old lady was there no dissenting vote on exten-

sive and slightly hazardous diagnostic procedures. She said that everything possible to keep her alive was to be done in order that she might follow interesting national political developments. She even exacted a promise from her physician that he become more knowledgeable by having an autopsy.

Sir George Pickering, Professor of Medicine at Oxford, recently stated in Texas that he would rather die in Great Britain than America. We have too many ways to keep patients alive. The author hopes to learn his views on the management of these two major disorders in elderly patients and to transmit them to the *Journal.*

SUMMARY

Gastrointestinal bleeding is a serious, and unfortunately not rare, complication of aortic stenosis. There is no agreement as to the mechanism of the bleeding. In spite of the precarious cardiac status of the patient the medical-surgical-radiological team is obligated to bend all efforts to locate the source of the bleeding. Attention to the upper gastrointestinal tract in all cases of cardiac decompensation is indicated.

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Project HOPE continues its programs in countries visited by the hospital ship S.S. HOPE: In a children's orthopedic rehabilitation center in Saigon, South Vietnam; in miniature HOPE's on the Amazon River in Peru and in medical schools and hospitals in South America and Africa.

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As of mid-1965, the American public had contributed $7 million in cash to Project HOPE, operators of the S.S. HOPE. Private industry has donated $7 million in supplies and equipment to the hospital ship; the pharmaceutical field alone has contributed $3 million in drugs.

Clinico-Pathological Exercise*

Presented by Rudolf Eyerer, M.D.**

Clinical Discussion by A. Physician, M.D. (anonymous)

Clinical Abstract

This 59-year-old white female was admitted to the hospital with a history of vomiting and visual disturbances. Up until two months prior to admission she was in her usual good health. At that time experienced loose bowel movements and vomiting of two days' duration. Her loose stools ceased but she continued to vomit at irregular intervals especially after being upset emotionally. This might occur immediately after a meal or about one half hour after a meal. There was no hematemesis or abdominal pain. Slight constipation seemed to follow the two brief episodes of loose stools. Prior to the onset of this illness she had been on a diet for obesity, diabetes and hypertension and had lost approximately thirty pounds.

At or about the time of her gastrointestinal upset the patient developed diplopia and blurring of vision. This was followed by photophobia and increased tearing but without apparent conjunctivitis. There has also been some difficulty in raising her eyelids, especially on the left. She has never had previous ophthalmalogic problems and has never worn glasses.

She is known to have had diabetes for approximately nine months and has been well controlled by diet alone.

For many years she has been hypertensive. There is no history of convulsions, headaches or vertigo. There has been slight diminution in her hearing for several years. Since the onset of her present illness there has been some dizziness with some tending to tip to one side or the other when she tried to sit up straight. At such times she feels as though her head were trembling.

About seven years ago she reached the menopause, with subsequent bleeding, spotting and vaginal discharge.

Physical examination reveals a well-developed, well-nourished woman in no acute distress. Temperature 100°F.; pulse 84/min.; respirations 60/min.; and blood pressure 150/100 mm.Hg. There is bilateral ptosis of the lids, more marked on the left. There is inability to move either eye medially or laterally but there is no apparent limitation of upward or downward gaze. The pupils are round, regular and equal and they respond to light. The fundi appear normal. The ear canals and tympanic membranes are not remarkable. The thyroid is not enlarged. There is no demonstrable cervical lymphadenopathy. The breasts are pendulous without masses or discharge from the nipples. There is a freely movable, rubbery, non-tender mass in the right axilla measuring 6 x 7 cms. The lungs are clear to auscultation and percussion. There is no cardiomegaly, murmur, thrill or friction rub. The abdomen is soft without palpable masses. The cervix appears grossly normal and cytology shows no tumor cells. The deep tendon reflexes are brisk and symmetrical. Pain, vibratory and position sense are intact. The left finger to nose test is poorly performed. The right finger to nose test is normal. The Romberg test is not done because of weakness of the patient. There is right facial paresis. There is normal corneal sensation. There is no nystagmus. The patient is well oriented. There is no aphasia. No pathologic reflexes can be demonstrated.

Laboratory findings include Hb. 14.5 gm/100 ml.; WBC 7,600/mm^3.; negative stools (x3) for occult blood; blood glucose 84 mg/100ml.; BUN 19 mg/100ml; VDRL negative; and a negative urine culture. Microscopic examination of the urinary sediment showed rare hyaline casts and a few leukocytes.

Course in Hospital:

The right axillary lymph node was removed and showed a metastatic, grade II, epidermoid carcinoma. No primary source for this tumor was found. A trial test of prostigmine showed no improvement in her neurological findings. She appeared to become more lethargic while in the hospital, where she remained for twelve days. Two days after she returned home at about 3:00 A.M. she suddenly developed a cough accompanied by rapid and noisy breathing. She was readmitted to the hospital showing much the same findings she had shown at the time of discharge two days previously. She was somewhat more lethargic and her speech was slow and thick. A spinal tap revealed one leukocyte and twelve erythrocytes per cu.mm. The spinal fluid protein was 76 mg/100 ml. Cytology of the spinal fluid showed a few suspicious cells (Class III). The patient expired on the second day following her readmission.

Ode To An Undiagnosed Mortal

Breathes there a Doc with soul so dead – who never to himself has said
At autopsy table to pathologist-friend – I knew all along this was how it would end
The case I am pondering and presenting today – Like many before it had ended that way
A complex of symptoms, opinions and work – Was greeted by Rudy with nary a smirk

*From the Medical and Laboratory Services, Eastern Maine General Hospital, Bangor, Maine.

**Assistant Pathologist, Eastern Maine General Hospital, Bangor, Maine.

If an answer from him I'd ever hoped to find – I most certainly must have been out of my mind
Each organ removed with near surgical skill – Served only to increase her hospital bill
For from gross pathological studies there came – Not an answer but rather this CPC game
She's been vomiting for a month said the nurse on the ward – When she called to report a new patient aboard
And my history would prove these facts were true – She did without warning and after meals too
No pain did she suffer or intolerance to food – And occasionally would upchuck in a nervous mood
Her weight loss was considerable 20 lbs. in 30 days – And another 30 lbs. or so back along a ways
An associated problem was some trouble with her vision – first some haziness and later single objects in divisions
No discomfort did she notice though light did make her blink – and lifting lids was difficult, the rt. one worse I think
She had no previous history of visual afflictions – But more were in store was my original prediction
She also had some sugar in her urine back along – But her diet alone had corrected this wrong
Her blood pressure problem of 8 months' duration – Had been treated at times with drugs for sedation
She denied all the usual symptoms and signs – Of angina, failure & her wind was just fine
Past and family history according to her story – Yielded nothing at all helpful, pertinent or gory
A systems review revealed the following clues – Whether helpful or not I shall later have to choose
Slightly decreased hearing and dizziness when upright – And painless trembling in her head on me had shed no light
Endocrine and respiratory systems functioned well – No urinary symptoms or post-menopausal spells
So now let's proceed from history in turn – To physical findings from stem to stern
Tachypnea, fever, and slight hypertension – Head normocephalic, hardly worthy of mention
The eyes quite the opposite demand close inspection – Bilateral ptosis beyond any correction
Extraocular movements can best be described – As slight up and down but none side-to-side
Pupillary movements were thought quite correct – Fundoscopic exam no lesions did detect
Ears, nose and throat, neck, lungs and heart – Seemed O.K. to me though I'm not always smart
Breasts were normal though a right axillary lump – Arose to inspection, rubbery, moveable, non-tender and plump
Abdominal palpation yielded no new clues I'm told – but perineal tumor stood out soft, long and bold
This obvious lesion historically had been noted – Following a pregnancy when her belly wasn't bloated
Pelvic exam yielded nothing it's said – As best can be told on Ward R in a bed
Neurological testing the night of her admission – Showed nothing else important relative to her condition
And now with the physical out of the way – Let's look at the lab data and see what they say
CBC and urine were normal but for pus cells – Clean voided culture showed all was quite well
BUN, serology, FBS all in line – Stools for occult blood and ECG fine
Skull x-rays taken and chest x-ray too – Showed nothing important or I missed the clue
Axillary biopsy was next on the list – Obscure diagnoses Dr. Eyerer seldom missed
Epidermoid carcinoma was his pontifical decision – But he could only guess from where it had arisen
Myasthenia? you are wondering neuromotor fanatics – Prostigmine was given, the results not dramatic
CSF studies with protein elevation – And class III cytology met with elation
Might have made *her* quite healthy, me wealthy and wise – But these facts were reported only after her demise
Consultants aplenty were called in to see – This interesting problem that was puzzling me
Gave several quite different and new points of view – She seemed in the long run to puzzle them too
So to autopsy table we went with great gusto – But the gross pathological facts were a busto
So I walked from the morgue with my head hanging low – Assured what the cause was I never would know
And that was the story when Rudy said to me – I'd like you to discuss a great CPC
Now you're probably wondering just what are my thoughts – At this stage of the game have I discovered the plot
Complete ophthalmoplegia affecting both her eyes – Most certainly in the 3rd nucleus lies
What agents could do this, I thought perhaps maybe – A stroke, diabetes or perhaps even tabes
But a vascular lesion confined such as this – if ever described the textbooks had missed
A normal blood sugar and negative serology – Ruled the latter two out without further apologies
Myasthenia gravis a consultant thought best – I thought we'd ruled out by the prostigmine test
My concern focused then on lesions metastatic – And the biopsy report made me really ecstatic
But could wandering tumor cells choose such a small locus – The unlikelihood of this made me recheck my focus
When what to my wandering eyes should appear – But a case seen in JAMA early last year
That it had happened in only *one* case – Gave it the aroma and CPC taste

That I had seen the midbrain cut – perhaps should be discouraging, but
Rudy isn't beneath going back to the brain – And cutting and cutting and cutting again
So my first diagnosis of which I'm quite fond – Is tumor metastatic to the depths of the pons
An alternative I like at least at this moment is – Neuropathy, ocular etiology carcinomatous
Described at least once in the books that I read – Are the same eye findings but no tumor in the head
Viruses and toxins have been implicated – when the tumor in some other site is located
Most frequently mentioned are the breasts and the lungs – But perhaps in this case in the uterus is hung
So I'll go down swinging on these two alternatives – And leave it to Rudy to list the superlatives

Clinical Impression

Intracranial lesion of uncertain etiology
Question of metastatic tumor (primary site unknown)
Mild diabetes. Hypertension

Poetic Conclusions

Neuropathy of viral or toxic etiology in patient with obscure carcinoma
Metastatic carcinoma of pons (primary tumor obscure)

Final Gross Anatomical Diagnoses

Encephalomyocarditis of viral origin
Bilateral pulmonary edema
Recent pulmonary embolus
Cholelithiasis
Chronic, bilateral, pyelonephritis
Arteriolonephrosclerosis, bilateral

Postmortem Findings — Dr. Rudolf Eyerer:

At autopsy the pupils were of unequal size, the right measuring 0.3 cm., the left 0.5 cm. in diameter. There was hypertrichosis of the upper lip and chin. Multiple sections through the breasts showed no evidence of neoplasm. The peritoneal cavity contained no free fluid. Some free fluid was demonstrable in each pleural cavity and in the pericardial sac. The heart weighed 360 gms. The valves, myocardium and chambers were not remarkable. A small pulmonary embolus and a small area of recent infarction were demonstrable in the right lower lobe. Both lungs showed slight to moderate pulmonary congestion and edema. The spleen weighed 154 gms. The liver weighed 1414 gms. Neither of these organs showed pathological alterations. The lumen of the gallbladder was filled with stones measuring up to 1 cm. in diameter. The extra-hepatic bile ducts were clear. The pancreas showed no abnormalities. The distal esophagus showed areas of superficial mucosal necrosis. The stomach, small bowel, appendix and colon showed no pathology. Mesenteric lymph nodes were not remarkable. The adrenals were of normal appearance. The combined weight of the kidneys was 240 gms. The cortical surfaces were pitted. Sections of the kidneys showed the cortex to be irregularly variable in thickness. The right ureter was slightly dilated but otherwise appeared normal. There was a small intramural fibroid at the uterine fundus. Otherwise uterus and cervix were not remarkable. Tubes and ovaries were somewhat atrophic. The thyroid gland was coarsely nodular but otherwise unremarkable. No retroperitoneal lymphadenopathy could be demonstrated. No extradural, subdural or subarachnoid hemorrhage could be demonstrated in the cranium. The cerebral hemispheres were symmetrical and showed no abnormalities. The vessels of the Circle of Willis were well preserved. A pressure cone was demonstrable on the inferior surface of the cerebellum. Multiple sections through the cerebrum, cerebellum, mid-brain, pons, medulla and cervical spinal cord showed no demonstrable lesions.

No lesion could be found which might represent the primary site of the epidermoid carcinoma demonstrated in the right axillary lymph node.

Postmortem Findings (microscopic):

The major microscopic lesions were found in the brain and heart which eventually established the final diagnosis. The myocardium showed a diffuse acute inflammatory infiltrate intermixed with a number of mononuclear cells. This infiltrate was accompanied by a fair amount of interstitial serous exudate. As an older underlying process, we found irregular areas of fibrosis separating the muscle bundles. The muscle bundles were irregular in size and many of the nuclei revealed enlargement and hyperchromasia. Irregular fibrous thickening of the endocardium were evident as well as sclerosis of the small sized arteries.

The cerebral lesions were quite striking and involved rather extensively both hemispheres, pons, medulla and cerebellum. A marked mononuclear cuffing was present in all these areas and was also seen about the vessels in the Robin-Virchow spaces. The ependyma in the third and fourth ventricle was partially effaced and the immediate adjacent brain substance revealed acute as well as mononuclear infiltrate. Satellitosis was present and focal pallor of the brain substance, as evidence of cerebral edema, was seen. All three were noted not only in the cortical areas but also in the basal ganglia and in the dentate nucleus in the cerebellum as well.

The inflammatory changes in the liver blend into the disease process rather well and present a diffuse acute inflammatory infiltrate with marked central necrosis. Focal bronchopneumonia, acute bronchitis and pulmonary edema represent an accompanying but more terminal process while the findings of chronic pyelonephritis, chronic cholecystitis and cholelithiasis seem to represent older non-related findings.

In conclusion the final pathologic diagnosis is:

Encephalomyocarditis of viral origin

REFERENCE

Viral and Rickettsial Infection of Man by Thomas M. Rivers, M.D., Frank L. Horsefall, M.D., J. B. Lippincott Company.

489 State Street, Bangor, Maine

Editorial

That Extra Effort

The process of medical education has increased in cost to the level where the students' share is more than most medical students and their families can readily absorb and yet, the students' share is still only a part of the total cost of his medical education. The University's financial share is "the greater half."

The latest charts of the Association of American Medical Colleges show the total (per student) cost of educating a medical student is above $4500.00 and the tuition in most medical schools is less than one half of this figure. Another A.A.M.C. chart shows what we all must have suspected—that medical students come mostly from the families in the upper middle and high income groups. If we are obtaining from these groups all the medical students we can, then our search for able candidates should be expanded in the lower middle and lower income groups.

For the State of Maine, these considerations hold a special significance. We have fewer high school students going on to college and fewer college students going on to graduate schools than most states. And, there is a well documented, state-wide shortage of practicing physicians. The possibility of a medical school for Maine is being studied with the hope of producing more doctors for the State. But, if we had a medical school in Maine tomorrow, where would the students come from? Our total production of medical students in the past ten years would barely fill half the school.

While we are waiting for the medical school for Maine, the time can be well used to encourage the able students of our secondary schools to go to college with the specific goals of graduate medical education and medical practice in Maine.

Recruitment, Aid and Placement is the name of the Maine Medical Association's Committee which has, over the past few years, been working on the problem. The Committee is to be commended for its efforts, but it will take all of the membership working together to solve this one. A new year is coming and this could be your most important resolution.

DEAN H. FISHER. M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

New Functions For Welfare Under Medicare

(Ed. Note: The following information is reprinted from a recent issue of the Department's Monthly Newsletter)

Maine is one of the few states in the union which combines under one administrative head the subjects of both Health and Welfare. For this reason Maine is well postured for the current impact nationally which is precipitating the union of health and welfare in retooling services to meet the requirements of the Social Security Amendments of 1965.

The Welfare Administration at the national level — which reflects similar responsibilities at the State level — "will be responsible for the development and administration of health and medical care standards for State programs for needy and near-needy individuals and families as well as for administration of project grants for comprehensive health programs for school and preschool children. All four programs — hospital insurance, supplemental medical insurance for the aged, the health and medical care aspects of our public assistance programs, and the child welfare programs — will be carefully coordinated so that each will serve its intended purpose. In carrying out their various responsibilities under the new legislation, both the Social Security Administration and the Welfare Administration will work very closely with the Public Health Service, which will have the principal responsibility for the professional aspects of the hospital and medical insurance programs."

Health Care For Low Income Families

The most far reaching provisions of this new legislation are those designed to bring the benefits of modern health and medical care to all needy people — recognizing that poor health is a major cause of poverty and other costly social problems. The law does this by authorizing Federal aid to States to enable them, at their option, to take major steps to implement the health care programs.

Impact Of Title XIX

This is a new Medical Assistance program that would ultimately include most of the medically indigent developed in stages.

1. First priority is to those already receiving public assistance. Benefits afforded this group must not be less than those provided others listed below, but may be greater in scope.

2. In second order of priority states may include those aged, blind, disabled, and children from broken homes whose income disqualifies them for the state aid programs but is insufficient to meet medical costs.

3. The third group that may be covered and perhaps the most extensive and far-reaching is children under 21 whose families indicate they cannot pay for the child's medical care.

States may purchase benefits directly from providers of services as most public welfare agencies in the country are now doing, or under the new Act, states may "buy in" to the Social Security program and provide physician services under Part B of Title XVIII. If this is done, states must pay the deductibles, the co-insurance costs and the so-called "20%" not covered under the insurance programs under both the hospital or supplementary benefits.

Under the new program the medical care available to all persons whom it includes, irrespective of age, must be comparable in amount, duration, and scope.

No length of residence requirement can be set for people who are included in the program and the only relatives who can be required to contribute are a spouse or the parents of a minor child or a disabled child of any age.

Effective Dates — The new Medical Assistance program will go into effect, on an optional basis with the States, on January 1, 1966. If States are to continue medical assistance for any group, including that now carried under the State aid programs, they will need to convert from existing programs to the new program by January 1, 1970, for the law now provides that Federal sharing in the costs of aid in medical or other remedial care will terminate under present programs.

Services Required — States coming into the new program before July 1, 1967, must provide some institutional and some non-institutional care for all recipients. However, after June 30, 1967, a State adopting the Title XIX program must provide inpatient hospital

Missing Pages ads

services, outpatient hospital services, other laboratory and x-ray services, skilled nursing home services for persons over 21 years of age, and physicians' services (whether furnished in the office, the patients' home, a hospital, nursing home or elsewhere). There must be comprehensive medical and dental coverage by 1975. The States are expected to set standards that will be appropriate to insure that the services will be of high quality and to adopt simplified methods of administration designed to assure that the services are furnished in a sympathetic, dignified and helpful manner.

State Planning — With the emphasis on quality of care in the new program, States will need to weigh carefully when they are ready to go into the program. The financial attractions are only one element. The new program will require substantial upgrading in scope and quality of services. Some new legislation and policy changes may be required as well as evaluation of fiscal capacities. All of the highly complex administrative factors must be assessed promptly so that realistic planning can be undertaken.

Increased Federal Matching Funds — In order to receive any additional Federal funds as a result of expenditures under the new program, present expenditures must be continued for all welfare programs combined at their present rate. With unreduced expenditures an increase in Federal participation in medical care may be expected. Compensation and training of professional medical personnel will bring a 75% Federal share as compared with the 50-50 Federal-State sharing for other administrative expenses.

Other Improvements In The Public Assistance Programs

The new law attacks one of the most fundamental problems in the public assistance programs — the low payments which tend to lock people into a life of extreme deprivation with little hope of escape. It does this in the following ways:

- *Work Incentives* (liberalized accounting of income and disregard of children's earnings)
- *Higher Payments* (revised Federal matching formulas)
- *Retention of Benefits* (disregarding lump sum Social Security benefits)
- *Payments for Students* (broadened to include students 18-21 who are attending a college or university)
- *Aid for Tuberculosis and Mental Patients in Institutions* (removes exclusion from Federal matching in OAA and MAA conditional upon maintenance of present State expenditures, improvement of mental health program and acquisition of new professionally trained public welfare staff)
- *Protective Payments for Third Persons* (on behalf of recipients in adult categories unable to manage their money because of physical or mental incapacity)
- *Alternative Formula for Federal Participation* (offers potential for substantial administrative simplification under Title XIX)

Special Programs For Children

The provisions in the new law which relate specifically to children are almost as far reaching in their potential benefits to the on-coming generations as are the insurance provisions of the elderly. In addition to the new medical assistance program which should, within 10 years at the most, assure high quality preventive, curative and remedial care to all medically needy children, there are additional provisions for child health and child welfare. These are the major ones:

1. *Maternal and Child Health, Crippled Children, and Child Welfare Services* — the law increases the amount authorized for child welfare services over current authorizations.
2. *Crippled Children-Training Personnel* — Major grants to institutions of higher learning for training professional personnel for health and related care of crippled children, particularly mentally retarded children and children with multiple handicaps.
3. *Health Care for Needy Children* — A five-year program of special project grants to provide comprehensive health care and services for children of school age, or for preschool children, particularly in areas with concentrations of low-income families. . . . Projects will provide screening, diagnosis, preventive services, treatment, correction of defects, and aftercare, including dental services, for children in low-income families.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

THE AMERICAN CANCER SOCIETY AND THE PHYSICIAN

John P. Wilson, M.D., *Atlanta*

THE AMERICAN CANCER SOCIETY, more so than some other voluntary health agencies, has a particularly close relationship with the medical profession. While it is, in large part, a voluntary lay organization, its direction and function is through the medical profession and it depends on the active participation of doctors in the various facets of its programs to carry out that function. It is well then for the medical profession to be familiar with these programs.

The American Cancer Society is engaged in three programs primarily: 1. Research. 2. Service To Patients. 3. Professional and Public Education. In each one of these there is an effective relationship with the medical profession.

Research

A portion of the funds of the American Cancer Society are allotted to direct support of research in cancer. There are, of course, several agencies involved in cancer research, including the Federal Government. However, the American Cancer Society supports a significant program of research and allows especially for latitude in areas which are not covered by other agencies. There are a number of research projects in Georgia which are supported by the American Cancer Society.

Patient Services

Because many of the needs of indigent patients are cared for by state and local programs, such as Cancer State Aid, the direct care of cancer patients is not a major portion of the American Cancer Society's program. It would financially be impossible for the Cancer Society to underwrite the care of any significant number of cancer patients. However, there are many areas in the direct care of patients in which the Cancer Society is active.

1. *Dressings and Loan Closets.* Many of the units of the American Cancer Society, particularly in the larger cities, have loan closets to provide beds, wheel chairs, crutches and sick room conveniences. Dressings are also prepared by volunteers and are available without cost to cancer patients.

2. *Transportation* may be provided for indigent patients to and from Cancer Clinics by volunteer workers, and in particular cases, by financial arrangements where other means of transportation are not available.

3. *Pain Relieving Drugs.* A limited allotment per month for advanced and terminal cancer patients is available for indigent patients.

4. *Information Center.* In some units of the Cancer Society, as in the Fulton County Unit, there is an Information Center to help indigent cancer patients contact and make arrangements with the appropriate agency for their needs. Contacts with Laryngectomy and Ileostomy Clubs can be arranged for rehabilitation of postoperative patients.

5. *Support of Institutions for terminal patients.* In the Atlanta area the Cancer Society contributes to the support of the Home of Our Lady of Perpetual Aid.

Professional and Public Education

The major activity of the American Cancer Society is the area of Professional and Public Education. While other agencies are involved in patient care and research, the American Cancer Society is the only agency which is engaged in public education regarding early diagnosis and treatment of cancer, which is, of course, basic in cancer control today.

1. Professional education includes an attempt to help physicians keep abreast of current information regarding cancer through the publication *Ca,* special bulletins and sponsorship of seminars and speakers at medical meetings. Professional films are also available for programs and meetings. Speakers can be provided for county society meetings.

2. The public education program of the American Cancer Society is designed to enlighten the public on the importance of early diagnosis and treatment for successful cancer control, through meetings, film showings, radio, newspaper and TV programs and special public educational publicity programs, such as the recent Pap smear program in Fulton County. An abundance of literature and a number of excellent films are available without cost.

Stress has been placed on the importance of early diagnosis by the seven danger signals and by advising Annual Health Examinations, including Pap smear and proctosigmoidoscopic examination. The importance of the prevention of cancer has been stressed in the antismoking campaign among teen-agers.

How Effective

How effective is the public education program and are doctors taking advantage of this program? In some areas there has apparently been good response. The effectiveness of the Pap smear has been apparent and it is the rare physician today who does not utilize this extremely effective diagnostic tool. On the other hand, routine proctosigmoidoscopic examination has not met with ready acceptance. Some reasons are obvious. It is more difficult, time consuming, requires preparation and is more uncomfortable than the Pap smear, and yet 75% of all cancer of the colon and rectum, the most common of all cancers, are located within reach of the sigmoidoscope. *There is no question that statistically there is a greater yield of treatable neoplasms as a result of routine proctosigmoidoscopic examination than with routine chest x-ray.*

It is important that the physician familiarize himself with the Cancer Society's program and to provide the patient with the basic cancer detection methods available, including the Annual Health Examination, Pap smear, proctosigmoidoscopic examination, instruction on breast self-examination, chest x-ray and other examinations as indicated. It is an uncomfortable question to have a layman ask occasionally, "What do I do when my doctor won't do a proctosigmoidoscopic examination or a Pap smear?"

The Cancer Society has a real service to offer the physician who will take advantage of it.

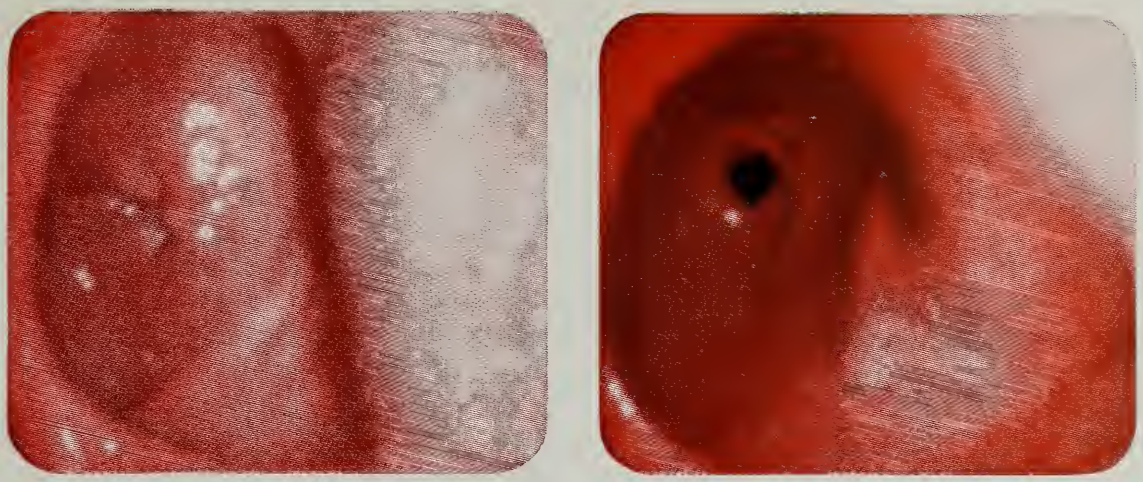
304 Boulevard, N.E.

Approved by the Professional Education Committee, Georgia Division, ACS.

Reprinted from The Journal of the Medical Association of Georgia, June, 1965, *Vol.* 54, *Page* 233.

New Drug Score: USA 75, USSR Zero

How important is the patent system to the prevailing health care picture? Here are a few examples. In 48 years, Russia's government-owned pharmaceutical industry has not developed a single new and important drug. In 24 years, thanks in part to patent protection, the American pharmaceutical industry has come up with at least 75 new drug entities. Credit (and a considerable amount is due) must go to a competitive industry that thrives in a competitive economy — motivated by the rewards and contributions to knowledge that the nation's patent system has always guaranteed. Editorial in **GP** (32:5), November 1965.



Intragastric photography studies[1]

A/ E. B., male, age 48. Normal antral contraction. Pyloric opening is not seen. It is difficult to differentiate a deep prepyloric contraction from a "pyloric fleurette" or true pylorus.

B/ Same subject after 6 mg. of propantheline bromide intravenously; antral contractions ceased. The pyloric orifice remained open and was easily identified. Better visualization of the antrum was also obtained.

Now you can see Pro-Banthine® at work
(propantheline bromide)

Pro-Banthine is so effective in anticholinergic action that it may be employed in visualizing the entire pyloric region.

In addition to the intragastric photographs, cinegastroscopic studies[2] have demonstrated graphically not only its effectiveness but the superiority of Pro-Banthine over belladonna alkaloids.

Pro-Banthine produced complete cessation of gastric, antral and pyloric motor activity with a dose of 6 mg. intravenously. This is approximately one-third the usual oral dose of 15 mg.

Atropine at full normal dosages did not produce such cessation. It required double the usual oral dose of atropine, 0.8 mg. intravenously, to duplicate the aperistaltic action of Pro-Banthine. This dose of atropine produced pronounced discomfort and tachycardia with ventricular rates as high as 150 per minute.

It is this pharmacologic superiority of Pro-Banthine which has made it the most widely prescribed anticholinergic in such conditions as peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Dosage—The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six 15 mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications—Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. The drug is contraindicated in patients with glaucoma or severe cardiac disease.

1. Barowsky, H.; Greene, L., and Bennett, R.: Investigators' Clinical Report. Photographs courtesy of Drs. H. Barowsky, L. Greene and R. Bennett.

2. Barowsky, H.; Greene, L., and Paulo, D.: Paper read at Meeting of American Society for Gastrointestinal Endoscopy, Montreal, Canada, May 25-27, 1965.

SEARLE

Research in the Service of Medicine

Maine Heart Association Notes

116 State Street, Augusta, Maine

Four Years' Experience With External Cardiac Resuscitation

"The principles of treatment of cardiac asystole or ventricular fibrillation by immediate thoracotomy for cardiac massage combined with adequate oxygenation are firmly established. When applied by experienced personnel in the operating room or postanesthetic recovery rooms, this technique has saved many lives. In other areas of the hospital and, particularly, outside of the hospital . . . thoracotomy for cardiac massage has rarely been successful.

"The introduction of an external technique for cardiac massage . . . in 1959 has led to a profound change in the prognosis of cardiac arrest . . . the results have been so uniformly superior to open cardiac massage that the latter is now used only when the chest is already open or where cardiac tamponade is suspected.

"External measures have certain definite advantages over direct cardiac massage . . . and can be successfully used in unusual situations where unplanned thoracotomy is best avoided or is impossible. Some of these latter situations include the lack of surgically trained or oriented personnel and lack of surgical instruments and respiratory equipment . . . It is clear from our past experience that the added trauma of opening the chest has rarely been tolerated by patients with unusually dilated or hypertrophied hearts, especially when the arrest has occurred in places where operative facilities were not available.

"Success depends on rapid recognition of the catastrophe followed by immediate rhythmical lower sternal compression preferably with the patient lying on an unyielding surface. Oxygenation is simultaneously carried out by mouth-to-mouth breathing or by the anesthesiologist if one is present.

"This experience has confirmed for us the value of a vigorous approach to cardiac resuscitation in all patients where death is considered to be premature and unexpected."

Submitted by Jacob B. Dana, M.D.

Reference: Lillehei, C. W. et al. JAMA, Volume 193, pages 651-658, 1965.

Letters To The Editor

To The Editor:

I believe the efforts of Dr. Rowland B. French in reporting the results of the "Office Evaluation of Streptococcal Infections of the Upper Respiratory Tract" which appeared in the October, 1965 issue of **The Journal of the Maine Medical Association,** is highly commendable. It must be called to your attention, however, that the interpretation of the diagnostic methodology is not correct and I believe the point of this investigation is entirely missed in the report. In the author's reference to the diagnosis of beta hemolytic streptococcal infections, he states the diagnosis was made by fluorescent antibody method at the diagnostic laboratory of the Department of Health and Welfare, Augusta, Maine, and the following results reproduced by their kind permission.

The fluorescent antibody method is one of the several methods for determining whether an organism is a member of Group A Lancefield beta hemolytic streptococci. If the author intended to call attention to Lancefield Group A, it has not been mentioned in the report but instead reference is only made to the determination of beta hemolytic streptococci which is still determined by observing the type of hemolysis on a blood agar plate. I believe the report would have much more significance if the author could clarify whether he is referring to beta hemolytic streptococci or Lancefield Group A Streptococci.

Joseph E. Porter, M.D.
Portland, Maine

To The Editor:

In his letter, Dr. Porter has made the very important point that it is the Lancefield Group A streptococcus that is being determined. Since it is this group that causes rheumatic fever, I am sure it is important to clarify that this is the group of bacteria involved in my article in the October, 1965 issue of **The Journal of the Maine Medical Association.**

As Doctor Porter points out, the fluorescent antibody method detects Lancefield Group A beta hemolytic streptococci and that this was the laboratory method used in this study.

Another point, which I believe might bear emphasis, is the need for adequate therapy. Not mentioned in the report, but I believe may be a fairly common mistake, is the inadequate treatment once the diagnosis is made. The American Heart Association pamphlet "Prevention of Rheumatic Fever" states that continued treatment is needed to be effective.

Rowland B. French, M.D.
Eastport, Maine

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County Society Notes

OXFORD

The annual meeting of the Oxford County Medical Society was held on October 6, 1965 at the Bethel Inn in Bethel, Maine. The following officers were elected for the coming year:

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SOMERSET

The Somerset County Medical Society held their fall meeting at Bemis Restaurant in Bingham, Maine on October 19, 1965.

A report by George E. Sullivan, M.D. on recent thoughts concerning Medicare was heard.

The society regrets the loss of Gretl J. Hoch, M.D. of Jackman who is relocating in Phillips, Maine. The society also expressed a desire to hold a future meeting with the local legal association in an attempt to rectify several of our mutual problems.

The program consisted of case reports by Drs. John P. Dow, H. Carl Amrein and W. Edward Jordan, Jr.

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Henry Pearson, M.D., Brownfield, November 30, 1965

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December, 1965

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Shields, Daniel R.	369 Main St., Lewiston
Spear, William	107 Main St., Lisbon Falls
Steele, Charles W.	472 Main St., Lewiston
Tardif, Lionel R.	300 Pine St., Lewiston
Taylor, Richard W.	St. Mary's Gen. Hosp., Lewiston
Tchao, Jou S.	82 Pine St., Lewiston
Thacher, Henry C.	117 Goff St., Auburn
Tibbetts, Otis B.	181 Gamage Ave., Auburn
Tousignant, Camille	111 Pine St., Lewiston
Turcotte, Richard W.	70 Pine St., Lewiston
Wakefield, Robert D.	St. Mary's Gen. Hosp., Lewiston
Webber, Wedgwood P.	299 Main St., Lewiston
Willie, James A.	111 Webster St., Lewiston
Young, E. Stanley	Poland Spring
Zanca, Ralph	86 Pine St., Lewiston

HONORARY

Buker, Edson B.	R.F.D. 3, Auburn
Gauvreau, Horace L.	82 Pine St., Lewiston
Goodwin, Ralph A., Sr.	56 Denison St., Auburn
Miller, Hudson R.	11 Turner St., Auburn
Pratt, Harold S.	Livermore Falls
Russell, Daniel F. D.	Leeds
Williams, James A.	40 Pleasant St., Mechanic Falls

SENIOR

Chenery, Frederick L., Jr.	Monmouth
Cignere, Enstache N.	90 Webster St., Lewiston
Sweatt, Linwood A.	48 Drummond St., Auburn

JUNIOR

Swett, Alfred E.	Maine Medical Ctr., Portland

AROOSTOOK COUNTY

President — R. Paul Johnson, M.D.
Secretary — George J. Harrison, M.D.
Treasurer — Clyde I. Swett, M.D.

ACTIVE

Albert, Joseph L.	4 Pleasant St., Fort Kent
Albert, Rodrigue J.	77 Main St., Fort Kent
Aungst, Melvin R.	Morneault Bldg., Fort Kent
Carton, Arthur K.	Market Square, Houlton
Chow, Alroy A.	P. O. Box 228, Ashland
Collins, H. Douglas	Caribou Clinic, Caribou
Donahue, Clement L.	22 Sweden St., Caribou
Donahue, Gerald H.	4 Station St., Presque Isle
Dunham, Marguerite C.	P.O. Box 748, Presque Isle
Etscovitz, Eli A.	Cary Mem. Hosp., Caribou
Giberson, Raymond G.	156A Academy St., Presque Isle
Gormley, Eugene G.	Market Square, Houlton
Gregory, Frederick J.	So. Main St., Caribou
Griffiths, Eugene B.	350 Main St., Presque Isle
Gureli, Necdet	Cary Mem. Hosp., Caribou
Harrison, George J.	Market Square, Houlton
Harvey, Thomas G.	59 Mayo St., Caribou
Hayward, I. Mead	So. Main St., Caribou
Helfrich, Harry M., Jr.	122 Academy St., Presque Isle
Higgins, George F.	122 Academy St., Presque Isle
Hogan, Chester F.	62 Main St., Houlton
Johnson, Gordon N.	P.O. Box 86, Houlton
Johnson, R. Paul	Main St., Fort Kent
Kimball, Herrick C.	P.O. Box 372, Fort Fairfield
Labbe, Onil B.	Van Buren
MacDonald, Lewis V. A.	Main St., Washburn
Madigan, John B.	Houlton
Nicholas, Eric F.	Mars Hill

O'Brien, William A.	Arthur R. Gould Mem. Hosp., Presque Isle
Ouellette, Benoit	77 Main St., Fort Kent
Page, Rosario A.	22 Sweden St., Caribou
Pendleton, Arthur D.	3 Green St., Fort Fairfield
Pines, Philip	Maine St., Limestone
Price, Richard D.	R.F.D. 2, Caribou
Proctor, Ray A.	Garden Circle, Caribou
Reynolds, Arthur P.	29 Second St., Presque Isle
Rideout, Samuel	Green St., Fort Fairfield
Sanfacon, Philip G.	Main St., Van Buren
Smith, Carroll H.	Box 785, Presque Isle
Smith, Margaret S.	Box 967, Presque Isle
Somerville, Robert B.	45 Hillside St., Presque Isle
Somerville, Wallace B.	Mars Hill
Swett, Clyde I.	18 Sherman St., Island Falls
Toussaint, Leonid G.	P.O. Box 9, Fort Kent
White, Leland M.	So. Main St., Caribou
Williams, Edward P.	72 Main St., Houlton
Wilson, G. Ivan	40 Court St., Houlton

SENIOR

Boone, Storer W.	429 Main St., Presque Isle
Brown, Stephen S.	Mars Hill
Faucher, Francois J.	Grand Isle
Kirk, William V.	Eagle Lake

AFFILIATE

Osborne, John R.	Narrows Pond Rd., Winthrop
Savage, Richard L.	4 Elm St., Fort Kent

JUNIOR

Burr, Charles G.	East Millinocket
Frenette, Francis F.	13479 Clifton Blvd., Lakewood 7, Ohio
Herson, Joseph H.	111 E. 88th St., New York, N. Y., 10028

SERVICE

Harvey, William C.	59 Mayo St., Caribou

CUMBERLAND COUNTY

President — Alvin A. Morrison, M.D.
Secretary-Treasurer — Stanley B. Sylvester, M.D.

ACTIVE

Adams, Marvin C.	25 Bramhall St., Portland
Agan, Robert W.	144 State St., Portland
Analis, Harry	3 Forest Pk., Portland
Andrews, Anneliese M.	Maine Medical Ctr., Portland
Ansell, Harvey B.	39 Deering St., Portland
Applin, Hilton H.	6 Cumberland St., Brunswick
Aranson, Albert	39 Deering St., Portland
Asali, Louis A.	29 Deering St., Portland
Asherman, Edward G.	131 Chadwick St., Portland
Bacastow, Merle S.	22 Bramhall St., Portland
Bachrach, Louis	16 Union St., Brunswick
Baldini, Elio	22 Bramhall St., Portland
Baldwin, Warren C.	42 Deering St., Portland
Barnes, Kirk K.	Baribeau Dr., Brunswick
Barron, Martin A., Jr.	37 Deering St., Portland
Bearor, Robert A.	Maine Medical Ctr., Portland
Bennet, Eben T.	49 Deering St., Portland
Bettle, Ronald A.	32 Federal St., Brunswick
Betts, Anthony	Maine Medical Ctr., Portland
Bidwell, Robinson L.	31 Bramhall St., Portland
Bischoffberger, John M.	Naples
Bishop, Lloyd W.	211 Vaughan St., Portland
Blumberg, Edward	6316 Strickland Ave., Brooklyn 34, N. Y.
Bonney, James H.	229 Vaughan St., Portland
Bove, Louis G.	12 Deering St., Portland
Bowman, Peter W.	Box C, Pownal
Branson, Sidney R.	37 Main St., So. Windham
Briggs, Winton	507 Ocean St., South Portland
Brinkman, Carl A.	50A Flaggy Meadow Rd., Gorham
Broggi, Frank S.	18 Neal St., Portland
Brown, Douglas H.	548 Shore Rd., Cape Elizabeth
Bullington, Sunny J.	32 Federal St., Brunswick
Burnett, Claude A., Jr.	59 Deering St., Portland
Burnham, Harold N.	130 Main St., Gorham
Burns, Robert M.	P.O. Box 151, Westbrook
Burrage, William C.	57 Deering St., Portland
Capron, Charles W.	22 Bramhall St., Portland
Carson, Robert S.	Baribeau Dr., Brunswick
Chatterjee, Manu	Baribeau Dr., Brunswick
Christensen, Harry E.	South Freeport
Ciampi, Louis A.	326 Stevens Ave., Portland
Clark, Frederick B.	131 State St., Portland
Clark, Richard I.	Freeport Medical Ctr., Freeport
Clarkin, Charles P.	64 Brookside Rd., Portland
Cole, Donald P.	45 Deering St., Portland
Contartese, Michael	149 Main St., Freeport
Crane, Lawrence	157 Pine St., Portland
Cummings, George O., Jr.	47 Deering St., Portland
Cunningham, Alice N.	32 Federal St., Brunswick
D'Andrea, Anthony L.	131 State St., Portland
Daniels, Donald H.	R.R. No. 1, Readfield
Darby, Alfred E., Jr.	19 Deering St., Portland
Davidson, David	235 State St., Portland
Davidson, Gisela K.	235 State St., Portland
Davies, Lloyd G.	78 Main St., Fryeburg
Davis, Paul V.	94 Main St., Bridgton
Derry, G. Hermann	690 Congress St., Portland
Dienst, Stanley G.	190 Pine St., Portland
Dionne, Maurice J.	Baribeau Dr., Brunswick
Doby, Tibor	131 State St., Portland
Dooley, Francis M.	53 Deering St., Portland
Dore, Kenneth E.	133 Main St., Fryeburg
Douphinett, Otis J.	763 Congress St., Portland
Drake, Emerson H.	18 Bramhall St., Portland
Drexler, James E.	Ward Town Rd., Freeport
Dunham, Carl E.	188 State St., Portland
Dyhrberg, Norman E.	323 Main St., Cumberland Mills
Earnhardt, Joseph B.	55 Strondwater St., Westbrook
Eppinger, Ernst	52 Belmont St., Portland
Fagone, Francis A.	312 Congress St., Portland
Ferguson, Franklin F.	22 Bramhall St., Portland
Fife, James L.	Baribeau Dr., Brunswick
Finks, Henry B.	73 Deering St., Portland
Fish, Nicholas	235 State St., Portland
Fox, Francis H.	83 West St., Portland
Galen, Robert S.	6 Breckan Rd., Brunswick
Galindo, Jose H.	Box C, Pownal
Geer, Charles R.	208 Vaughan St., Portland
Geer, George I., Jr.	208 Vaughan St., Portland
Geyerhahn, George	73 Deering St., Portland
Gibbons, John F.	22 Bramhall St., Portland
Givertz, Bernard	131 Chadwick St., Portland
Glassmire, Charles R.	37 Deering St., Portland
Godsoe, John A.	7 Bramhall St., Portland
Goduti, Richard J.	9 Deering St., Portland
Good, Philip G.	38 Deering St., Portland
Greco, Edward A.	12 Pine St., Portland
Grish, Albert J.	Kings Park State Hosp., Kings Park, N. Y.
Hallett, George W., Jr.	72 West St., Portland
Handanos, Vassilios	Box C, Pownal
Hanley, Daniel F.	P.O. Box 637, Brunswick
Hawkes, Richard S.	47 Deering St., Portland
Heifetz, Ralph	173 State St., Portland
Herrick, Stanley E., Jr.	Maine Medical Ctr., Portland
Hiebert, Clement A.	18 Bramhall St., Portland
Hill, Douglas R.	855 Sawyer St., So. Portland
Hinckley, Harris	331 Cottage Rd., So. Portland
Holt, C. Lawrence	27 Deering St., Portland
Huntress, Roderick L.	988 Sawyer St., South Portland
Hurwitz, Alfred	Mt. Sinai Hosp., Miami Beach, Fla.
Jacobson, Payson B.	295 Brighton Ave., Portland
Johnson, Albert C.	131 Chadwick St., Portland
Johnson, Oscar R.	18 Deering St., Portland
Kent, Stanley W.	42 Deering St., Portland
Knowles, Robert M.	49 Deering St., Portland
Kunkle, E. Charles	131 Chadwick St., Portland
Lape, C. Philip	7 Bramhall St., Portland
Lappin, John J.	171 State St., Portland
Laughlin, K. Alexander	201 State St., Portland
Leary, Gerald C.	144 State St., Portland
Leighton, Wilbur F.	192 State St., Portland
Leiter, Laban W.	175 Vaughan St., Portland

Levy, Richard A.	Maine Medical Ctr., Portland
Libby, Harold E.	702 Main St., Westbrook
Lincoln, John R.	22 Bramhall St., Portland
Llorente, Aldo F.	56 Baribeau Dr., Brunswick
Lombard, Reginald T.	793 Main St., South Portland
Lorimer, Robert V.	169 State St., Portland
Love, Robert B.	97 Main St., Gorham
Lovely, David K.	46 Deering St., Portland
Lutes, Chris A.	7 Bramhall St., Portland
Mack, Francis X.	144 State St., Portland
MacVane, William L., Jr.	211 State St., Portland
Maier, Paul	723 Congress St., Portland
Maltby, George L.	31 Bramhall St., Portland
Marshall, Donald F.	25 Bramhall St., Portland
Marshall, Richard A.	22 Bramhall St., Portland
Marston, Paul C.	Kezar Falls
Martin, Ralf	131 Chadwick St., Portland
Martin, Thomas A.	157 Pine St., Portland
Matthews, Edward C.	131 Chadwick St., Portland
Mazzone, Giovanni	487 Stevens Ave., Portland
McAdams, William R.	723 Congress St., Portland
McAfee, Robert E.	18 Bramhall St., Portland
McCann, Eugene C.	49 Deering St., Portland
McCrum, Philip H.	188 State St., Portland
McFarland, Edward A.	Baribeau Dr., Brunswick
McIntire, Barron F., Jr.	13 W. Elm St., Yarmouth
McLean, E. Allan	29 Deering St., Portland
McManamy, Eugene P.	72 West St., Portland
McMichael, Morton	73 Deering St., Portland
Melkis, Andrew	P. O. Box 161, Gray
Miller, Thor	752 Main St., Westbrook
Mohlar, Robert G.	Baribeau Dr., Brunswick
Monaghan, Stephen E.	7 Bramhall St., Portland
Monkhouse, William A.	131 State St., Portland
Morrison, Alvin A.	57 Deering St., Portland
Morrison, Robert M.	148 State St., Portland
Moulton, Albert W., Jr.	180 State St., Portland
Moulton, Sumner E.	12 Deering St., Portland
O'Donnell, Eugene E.	32 Deering St., Portland
Olmsted, Burton L.	73 Deering St., Portland
Orbeton, Everett A.	131 Chadwick St., Portland
Osher, Harold L.	131 Chadwick St., Portland
Ottum, Alvin E.	148 State St., Portland
Pawle, Robert H.	8 Walcott Ave., Falmouth
Pennoyer, Douglass C.	112 Vaughan St., Portland
Penta, Walter E.	316 Woodford St., Portland
Perkins, Niles L.	16 Bramhall St., Portland
Pogue, Jackson S.	529 Gilmore Ave., Trafford, Pa. 15085
Poliner, Irving J.	235 State St., Portland
Polisner, Saul R.	143 Vaughan St., Portland
Porter, Joseph E.	22 Bramhall St., Portland
Proudian, Paul O.	776 Main St., Westbrook
Ray, Ferris S.	7 Bramhall St., Portland
Richards, A. Dewey	11 Gage St., Bridgton
Robinson, Hugh P.	27 Deering St., Portland
Rogers, Albert M.	157 Pine St., Portland
Rubins, Nina B.	E. A. Center Mem. Clinic, Steep Falls
Rubins, Talivaldis	E. A. Center Mem. Clinic, Steep Falls
Sager, George F.	18 Bramhall St., Portland
Santoro, Domenico A.	43 Deering St., Portland
Sapiro, Howard M.	171 State St., Portland
Saunders, Norman W.	12 Deering St., Portland
Sawyer, Howard P., Jr.	22 Bramhall St., Portland
Selvage, Irving L., Jr.	22 Bramhall St., Portland
Shapiro, Morrill	29 Deering St., Portland
Sirodot, George	Jackman
Skillin, Charles E.	690 Congress St., Portland
Skillin, Frederick W.	69 So. High St., Bridgton
Stevens, Theodore M.	148 State St., Portland
Strach, Toffield B. J.	3 Deering St., Portland
Sulkowitch, Hirsh	Box 1744, Portland
Sylvester, Stanley B.	Union Mutual Life Ins. Co., 400 Congress St., Portland
Tabachnick, Henry M.	110 Park Ave., Portland
Taxiarchis, Louis N.	144 State St., Portland
Taylor, William F.	131 U. S. Route 1, Falmouth
Telfeian, Alphonse	131 Chadwick St., Portland
Tetreau, William J.	144 Spring St., Portland
Thompson, Philip P., Jr.	131 Chadwick St., Portland
Toner, Cornelius A.	31 Bramhall St., Portland
Tougas, Raymond A.	Baribeau Dr., Brunswick
Turcotte, Guy N.	7 Bramhall St., Portland
Turgeon, Raphael F.	367 Main St., Westbrook
Turnbull, Elliott D.	Elm House, Naples
Urjanis, Janis	Box C, Pownal
Vanadzin, Boris A.	389 Congress St., Portland
Van Deventer, Wilhelm H. J.	289 Maine St., Brunswick
Van Lonkhuyzen, Maurice	131 State St., Portland
Ward, John V.	131 State St., Portland
Weaver, Michael L.	36 Federal St., Brunswick
White, William J.	1 Mitchell Rd., South Portland
Whittier, Alice A. S.	143 Neal St., Portland
Wight, Donald G.	30 Mitchell Rd., South Portland
Winkelbauer, Rudolf G.	Baribeau Dr., Brunswick
Woodman, Arthur B.	15 Johnson Rd., Falmouth Foreside
Wyman, David S.	47 Deering St., Portland
Zolov, Benjamin	296 Congress St., Portland

HONORARY

Barker, Nathaniel B. T.	1 South St., Yarmouth
Blaisdell, Elton R.	12 Deering St., Portland
Fogg, C. Eugene	35 Deering St., Portland
Foster, Thomas A.	131 State St., Portland
Melnick, Jacob	261 Congress St., Portland
Moulton, Albert W., Sr.	180 State St., Portland
Patterson, James	1 Bay Rd., South Portland
Ridlon, Joseph R.	58 South St., Gorham
Sowles, Horace K.	413 Blackstrap Rd., Falmouth
Thaxter, Langdon T.	Cumberland Foreside, Portland 200

SENIOR

Babalian, Leon	645 Congress St., Portland
Casey, William L.	131 State St., Portland
Cummings, George O., Sr.	47 Deering St., Portland
Freeman, William E.	107 Main St., Yarmouth
Petterson, Herman C.	Chebeague Island
Scolten, Adrian H.	32 Deering St., Portland
Webber, Isaac M.	29 Deering St., Portland

AFFILIATE

Getchell, Ralph A.	14 Elmwood Rd., Cape Elizabeth
Sidwell-Thompson, Doris M.	R.F.D. Whittier Rd., W. Ossipee, N. H.
Storer, Daniel P.	108 Fessenden St., Portland
Titherington, John B.	97 Brook Rd., Falmouth
Wellington, J. Foster	396 Brighton Ave., Portland

JUNIOR

Allen, Donald E.	Sebago Lake
Fogg, Philip S., Jr.	39 Upper Loudon Rd., Loudonville, N. Y. 12211
Gates, Clifford W.	130 Main St., Gorham
Hudson, Henry A.	R.F.D. No. 1, West Bridgton
Westermeyer, Marion W.	25556 Lomas Verdes St., Loma Linda, Calif. 92354

SERVICE

Stephenson, Richard B.	Bldg. 1, Rm. 118, National Institutes of Health, Bethesda, Md. 20014

FRANKLIN COUNTY

President — Harry Brinkman, M.D.
Secretary-Treasurer — Maynard B. Colley, M.D.

ACTIVE

Bowne, Hays G.	9A Main St., Farmington
Brinkman, Harry	47 Perham St., Farmington
Brinkman, Paul A.	Farmington
Colley, Maynard B.	14 Main St., Farmington
Covert, Stanley B.	Kingfield
Duffy, Wallace H.	100 Main St., Farmington
Eastman, Charles W.	15 Millett St., Livermore Falls
Fiorica, Gaetano T.	12 Church St., Chisholm
Floyd, Paul E.	2 Middle St., Farmington
Pope, W. Dean	6 Pleasant St., Rangeley
Reed, James W.	18 Main St., Farmington
Rowe, Gunther H.	42 Main St., Livermore Falls
Zikel, Herbert M.	High St., Wilton

HONORARY

Pratt, George L.	7 Main St., Farmington

SENIOR

Weymouth, Currier C.	Eastmont Sq., Farmington

JUNIOR

Marsters, David W.	51 Benson St., Glen Ridge, N. J.

HANCOCK COUNTY

President — W. Edward Thegen, M.D.
Secretary-Treasurer — Russell G. Williamson, M.D.

ACTIVE

Bromley, William C.	Medical Ctr., Southwest Harbor
Brownlow, Bradley E.	Blue Hill Mem. Hosp., Blue Hill
Cameron, Dwight	12 Mt. Desert St., Bar Harbor
Coffin, Ernest L.	Northeast Harbor
Cooper, Llewellyn W.	194 Main St., Bar Harbor
Crowe, James H.	57 Main St., Ellsworth
Gray, Philip L.	Blue Hill
Hsu, Theodore S.	14 High St., Ellsworth
Joost, Arthur M., Jr.	P.O. Box 502, Bucksport
Knickerbocker, Charles H.	15 High St., Bar Harbor
Kopfmann, Harry	Deer Isle
LaCasce, Joseph H.	50 Union St., Ellsworth
Lambdin, Morris A.	Maine Coast Mem. Hosp., Ellsworth
Lane, Russell M.	P.O. Box "J", Blue Hill
Larrabee, Charles F.	48 Mt. Desert St., Bar Harbor
McIntyre, John D.	50 Union St., Ellsworth
O'Meara, Edward S.	12 West Maple St., Ellsworth
Pease, Horace B.	Maine Coast Mem. Hosp., Ellsworth
Russell, Robert F.	Penobscot
Stadler, Eliot T.	West Gouldsboro
Suyama, Eji	58 W. Main St., Ellsworth
Thegen, W. Edward	Elm St., Bucksport
Torrey, Marcus A.	75 State St., Ellsworth
Wilbur, Herbert T., Jr.	100 Main St., Southwest Harbor
Williamson, Elizabeth E.	Blue Hill
Williamson, Russell G.	Blue Hill Mem. Hosp., Blue Hill
Wilson, Robert D.	Mt. Desert Island Hosp., Bar Harbor

SENIOR

Babcock, Harold S.	Castine

SERVICE

Black, Paul E. (Capt.)	MC USN NAS, Lakehurst, N. J.
York, Elihu	USN Air Development Ctr., AMAL, Johnsville, Pa. 18974

KENNEBEC COUNTY

President — John D. Denison, M.D.
Secretary-Treasurer — Earle M. Davis, M.D.

ACTIVE

Ashley, Alta	11 Weston St., Augusta
Barnard, John M. H.	Doctors Park, 89 Hospital St., Augusta
Barron, Richard E.	Main St., Monmouth
Beckerman, Stanley C.	82 Elm St., Waterville
Bhatnagar, Hemendra N.	30 Elm St., Waterville
Bolduc, Jean L.	173 Main St., Waterville
Bourassa, Harvey J.	47 Elm St., Waterville
Brann, Henry A.	31 Western Ave., Augusta
Bull, Frank B.	Hasson St., Farmingdale
Callahan, Robert L.	105 Water St., Augusta
Canal, Ory D.	193 Cony St., Augusta
Castellanos, Jose	Augusta State Hosp., Augusta
Chamberlin, Richard T.	14 Gilman St., Waterville
Chasse, Richard L.	18 Park St., Waterville
Chen, Jen-Ti	Cherry Hill Terrace, Waterville
Cook, Aaron	23 High St., Waterville
Crawford, Joseph R.	105 Water St., Augusta
Cruickshank, Frank S., Jr.	5 Lee St., Winslow
Dachslager, Philip	8 Green St., Augusta
Darlington, Brinton T.	Doctors Park, 89 Hospital St., Augusta
Davis, Earle M.	2 School St., Waterville
Delphin, Jacques M.	Hudson River State Hosp., Poughkeepsie, N. Y.
Denison, John D.	105 Brunswick Ave., Gardiner
Dennis, Richard H.	33 College Ave., Waterville
Dole, Richard R.	218 Main St., Waterville
Dore, Clarence E.	2 School St., Waterville
Dunn, Robert H.	Veterans Adm., Togus
Emanuel, Meyer	Veterans Adm., Togus
Ervin, Edmund N.	2 School St., Waterville
Fisher, Dean H.	State House, Augusta
Fisher, Samson	15 College Ave., Waterville
Giddings, Lane	6 E. Chestnut St., Augusta
Giddings, Paul D.	31 Western Ave., Augusta
Giesen, Joseph H.	34 Gilman St., Waterville
Gingras, Adolphe J.	105 Water St., Augusta
Gingras, Napoleon J.	6 E. Chestnut St., Augusta
Goodof, Irving I.	Thayer Hospital, Waterville
Gould, George I.	79 Main St., Richmond
Guillemette, Maurice R.	109 Water St., Augusta
Guite, L. Armand, Sr.	45 Elm St., Waterville
Guite, L. Armand, Jr.	45 Elm St., Waterville
Harlow, Edwin W.	177 Main St., Waterville
Hiebel, Joseph J.	34 Gilman St., Waterville
Hill, Howard F.	33 College Ave., Waterville
Hill, Kevin	33 College Ave., Waterville
Hirschberger, Celia	Augusta State Hosp., Augusta
Hornberger, H. Richard	2 School St., Waterville
Hurd, Allan C.	Hasson St., Farmingdale
Jan, M. Rafiq	100 Church St., New York, N. Y. 10007
Jones, Paul A., Jr.	2 School St., Waterville
Kirkpatrick, Price A.	Thayer Hospital, Waterville
Landwehr, George R.	21 Western Ave., Augusta
Lansing, Peter F.	16 Macomber Ave., Augusta
Lepore, Anthony E.	128 Maine Ave., Gardiner
Lopez, Eduardo A.	33 College Ave., Waterville
Marshall, Joseph A.	177 Main St., Waterville
Marshall, Paul A.	27½ Johnson Hgts., Waterville
Mathews, Hugh J., Jr.	345 Water St., Gardiner
McLarn, William D.	222 Main St., Waterville
McLaughlin, Clarence R.	345 Water St., Gardiner
McLaughlin, Ivan E.	345 Water St., Gardiner
McQuillan, Arthur H.	177 Main St., Waterville
Melendy, Oakley A.	Doctors Park, 89 Hospital St., Augusta
Michaud, Joseph C.	160 Silver St., Waterville
Milliken, Howard H.	R #1 Pond Rd. (Manchester), Hallowell
Monsivais, Alfredo	1 Western Ave., Winthrop
Moore, Valentine J.	Thayer Hospital, Waterville
Morris, Craig W.	50 Bangor St., Augusta
Nikolaidis, Demitrios	Augusta Gen. Hosp., Augusta
O'Connor, Francis J.	4 Woodlawn St., Augusta
Ohler, Robert L.	Veterans Adm., Togus
Patterson, John C.	Box 724, State Hospital, Augusta
Peddie, Harry M. K.	Doctors Park, 89 Hospital St., Augusta
Pfeiffer, Paul H.	14 Gilman St., Waterville
Plimpton, Jay R.	283 Water St., Augusta
Pomerleau, Ovid F.	179 Main St., Waterville
Pomerleau, Rodolphe J. F.	27 Main St., Waterville
Poulin, Albert A.	Cherry Hill Dr., Waterville
Poulin, James E.	177 Main St., Waterville
Pratt, Loring W.	177 Main St., Waterville
Provost, Helen C.	48 Green St., Augusta
Provost, Pierre E.	48 Green St., Augusta
Reynolds, John F.	216 Main St., Waterville
Richards, Lee W., Jr.	21 Western Ave., Augusta
Robertson, George J.	171 Harrison Ave., Boston 11, Mass.
Rodriguez, Jose M.	109 Silver St., Waterville
Rohm, Walter	Box 724, State Hosp., Augusta
Runyon, William N.	283 Water St., Augusta
Russell, Theodore M.	20 Western Ave., Augusta
Saunders, Allen I.	Ferry Rd., R.F.D. 2, Augusta

Schmidt, Lorrimer M.	Veterans Adm., Togus
Schumacher, William E.	14 Westwood Rd., MD "B," Augusta
Schwarz, Harald J.	Seton Hospital, Waterville
Seligman, Morris J.	Veterans Adm., Togus
Senenky, Joseph P.	Augusta State Hosp., Augusta
Sewall, Kenneth W.	2 School St., Waterville
Shaw, John H.	131 Sewall St., Augusta
Shelton, M. Tieche	21 Western Ave., Augusta
Simpson, Margaret R.	2 Sea Barn Rd., Cape Elizabeth
Smith, Kenneth E.	Veterans Adm., Togus
Sommerfeld, Kurt A.	Veterans Adm., Togus
Southern, Edward M.	1379 Balfour Rd., Grosse Pointe 30, Mich.
Spellman, Francis A.	Veterans Adm., Togus
Stinchfield, Allan J.	P.O. Box 343, Augusta
Stocks, Joseph F.	67 Silver St., Waterville
Sturtevant, Vaughn R.	33 College Ave., Waterville
Towne, Charles E.	18 Common St., Waterville
Trembly, Bruce	33 College Ave., Waterville
Veilleux, Lucien F.	30 Elm St., Waterville
Willard, Harold N.	Thayer Hospital, Waterville
Wilson, Robert W.	Veterans Adm., Togus

HONORARY

Crawford, Albert S.	East Blue Hill
Crawford, J. Ramser	105 Water St., Augusta
Hill, Frederick T.	Thayer Hospital, Waterville
Kagan, Samuel H.	283 Water St., Augusta
Reynolds, Ralph L.	216 Main St., Waterville

SENIOR

Bauman, Clair S.	159 Silver St., Waterville
Breard, J. Alfred	15 Summer St., Waterville
Goodrich, Blynn O.	165 Main St., Waterville
Herring, Leon D.	Memorial Dr., Winthrop
Langer, Ella	2 Chapel St., Augusta
Marquardt, Matthias	109 Cony St., Augusta

AFFILIATE

Reel, John J.	59 So. Front St., Richmond
Sanders, Stephen W.	120 Main St., Winthrop
Sleeper, Francis H.	19 Columbia St., Augusta

JUNIOR

Dillihunt, Richard C.	Maine Medical Ctr., Portland

KNOX COUNTY

President — Frank W. Kibbe, M.D.
Secretary-Treasurer — Peter H. Holz, M.D.

ACTIVE

Alexander, Fay K.	Camden Com. Hosp., Camden
Apollonio, Howard L.	22 White St., Rockland
Brouwer, Johan	5 Beech St., Rockland
Dennison, Frederick C.	3 Gillchrest St., Thomaston
Earle, Ralph P.	Vinalhaven
Eddy, Robert H.	5 Beech St., Rockland
Fuller, Barbara L.	20 Chestnut St., Rockland
Hawkins, Donald B.	Atlantic Ave. & Sea St., Camden
Holz, Peter H.	51 Elm St., Camden
Howard, Emery B., Jr.	23A Summer St., Rockland
Hunter, Albert L.	Camden Com. Hosp., Camden
Kangas, Onni C.	417 Main St., Rockland
Kibbe, Frank W.	R.F.D., Lincolnville
Kim, Hakkew	33 Main St., Thomaston
King, Merrill J., Jr.	22 White St., Rockland
Lathbury, Vincent T.	Medical Arts Bldg., Rockland
Lawry, Oram R., Jr.	96 Limerock St., Rockland
Mann, David V.	22 White St., Rockland
McLellan, William A.	87 Chestnut St., Camden
Millington, Paul A.	44 Mountain St., Camden
Morse, Edward K.	22 White St., Rockland
Onat, Mustafa V.	St. George
Root, John A.	22 White St., Rockland
Soule, Gilmore W.	22 White St., Rockland
Tounge, Harry G., Jr.	12 Union St., Camden
Ward, William W.	P.O. Box 804, Rockland
Wasgatt, Wesley N.	41 Talbot Ave., Rockland
Waterman, Richard	Main St., Waldoboro
White, Henry O.	22 White St., Rockland
Worthing, Verla E.	Box A, Thomaston

HONORARY

Campbell, Fred G.	P.O. Box 484, Warren
Frost, Harold M.	Friendship
Hall, Walter D.	407 Main St., Rockland

SENIOR

Jameson, C. Harold	Medical Arts Bldg., Rockland
Loewenstein, George	Chebeague Island
Winter address —	Woodside Dr., Clearwater, Fla. 33516
Naumer, Harry A.	Land's End, Port Clyde
Platt, Anna	Beauchamp Rd., Rockport
Saunders, Sallie H.	R.F.D., Camden

AFFILIATE

Bean, Achsa M.	Star Route 22-282, Owl's Head
Stimson, Barbara B.	Star Route 22-282, Owl's Head

LINCOLN-SAGADAHOC COUNTY

President — Carl R. Griffin, Jr., M.D.
Secretary-Treasurer — George W. Bostwick, M.D.

ACTIVE

Abdalla, Abdalla Y.	19 Bedford St., Bath
Akar, Hamdi	17 Grove St., Bath
Andrews, John F.	20 West St., Boothbay Harbor
Belknap, Samuel L.	Damariscotta
Blackburn, Nelson P.	Bath Memorial Hosp., Bath
Bostwick, George W.	P.O. Box 388, Newcastle
Burden, Charles E.	1 North St., Bath
Conner, William W.	61 Atlantic Ave., Boothbay Harbor
Doble, Miriam	Sugarloaf Mt., Kingfield
Dorogi, Louis V.	Old Post Rd., Rt. 138, Bowdoinham
Dougherty, John F.	112 Front St., Bath
Fichtner, Paul A.	781 High St., Bath
Gregory, Philip O.	St. Andrews Hosp., Boothbay Harbor
Griffin, Carl R., Jr.	69 Townsend Ave., Boothbay Harbor
Kinder, Edward L., Jr.	1027 Washington St., Bath
Lenfest, Stanley R.	Waldoboro
McLaren, John J.	Baribeau Dr., Brunswick
Nichols, Arthur A.	Edgecomb
Oceretko, Arkadij	37 Court St., Bath
Powell, Ralph C.	Damariscotta
Proctor, Thomas E.	Boothbay Harbor
Smith, Jacob	118 Front St., Bath
Smith, Joseph I.	118 Front St., Bath
Stetkevych, Alexander G.	858 Washington St., Bath
Tracy, Mary J.	Bristol Rd., Damariscotta
Wilson, Harry M.	944 Middle St., Bath
Winchenbach, Francis A.	910 Washington St., Bath
Zeller, Alan W.	35 Main St., Damariscotta

HONORARY

Kershner, Warren E.	57 Green St., Bath
Morin, Harry F.	905 Middle St., Bath
Stetson, Rufus E.	Damariscotta

SENIOR

Dalrymple, Sidney C.	So. Great Rd., So. Lincoln, Mass.
Sherman, Fuller G.	Spruce Pt., Boothbay Harbor

AFFILIATE

Bachulus, John M.	P.O. Box 285, Bath
Hamilton, Virginia C.	South Harpswell

OXFORD COUNTY

President — Henry M. Howard, M.D.
Secretary-Treasurer — Ake Akerberg, M.D.

ACTIVE

Akerberg, Ake	2 East Main St., So. Paris
Aucoin, Peter B.	151 Franklin St., Rumford
Bean, H. Richard	241 Main St., Norway
Defoe, Garfield G.	Dixfield
Dixon, Walter G.	16 Deering St., Norway
Elsemore, Dexter E.	11 Main St., Dixfield
Harper, Harry L.	17 Main St., So. Paris
Hiebert, Joelle, C., Jr.	Box 148, Norway
Howard, Henry M.	105 Franklin St., Rumford
Jackson, Norman M.	151 Franklin St., Rumford
Martin, Joseph E.	35 Main St., Mexico
Moore, Beryl M.	High St., Oxford
Nangle, Thomas P.	West Paris
Oestrich, Alfred	89 Congress St., Rumford
Pena, Manuel G.	82 Maine Ave., Rumford
Phillips, David L.	82 Maine Ave., Rumford
Rowe, Linwood M.	Rumford Com. Hosp., Rumford
Royal, Albert P., Jr.	82 Maine Ave., Rumford
Smith, Charles M.	Main St., Dixfield
Young, John	Paradise St., Bethel

HONORARY

Adams, Lester	9 Knox St., Thomaston
Mills, Nathaniel	North Main St., Wolfeboro, N. H.
Stanwood, Harold W.	c/o Kimball Stanwood, York

SENIOR

Hubbard, Roswell E.	Waterford
Kay, Edwin	31 Frye St., Lewiston
MacDougall, James A.	303 Penobscot St., Rumford
Nelson, Chesley W.	121 Main St., Norway

SERVICE

Boynton, Willard H.	U.S. A.I.D., A.P.O. 271, New York, N. Y.

PENOBSCOT COUNTY

President — Richard T. Munce, M.D.
Secretary — Charles D. McEvoy, Jr., M.D.
Treasurer — Benjamin L. Shapero, M.D.

ACTIVE

Adams, Asa C.	68 Main St., Orono
Adams, Winford C.	588 N. Main St., Brewer
Babcock, Edward B.	431 State St., Bangor
Barrett, Robert J., Jr.	Cor. Union & James Sts., Bangor
Bjorn, John C.	Hampden Highlands
Blackwell, William M.	Millinocket Com. Hosp., Millinocket
Blaisdell, Carl E.	47 Broadway, Bangor
Blaisdell, William B., Jr.	47 Broadway, Bangor
Blinder, Philip	336 Mt. Hope Ave., Bangor
Bridges, Donald E.	336 Mt. Hope Ave., Bangor
Brito, Joseph S.	St. Joseph Hosp., Bangor
Brod, James J.	415 Broad St., Oneida, N. Y. 13421
Brown, Eugene E.	57 Summit Ave., Bangor
Brown, Lloyd	186 State St., Bangor
Burke, John E.	824 State St., Bangor
Burke, Paul W.	5 High St., Newport
Butler, Harry	77 Broadway, Bangor
Butterfield, Wilfred I.	119 Main St., Lincoln
Chase, George O.	Eastern Maine Gen. Hosp., Bangor
Chason, Sidney	128 Broadway, Bangor
Clement, James D., Jr.	77 Essex St., Bangor
Clough, Dexter J., 2nd	224 State St., Bangor
Cornell, Robert C.	336 Mt. Hope Ave., Bangor
Coulton, Donald	326 State St., Bangor
Cross, Harold D.	Main Rd.& Summer Sts.,Hampden Highlands
Curran, Edward L.	209 State St., Bangor
Cutler, Lawrence M.	31 Grove St., Bangor
Desjardins, Richard F.	240 Penobscot Ave., Millinocket
Dietrich, Mary M.	P.O. Box 93, Orrington
Duffey, Richard V.	187 N. Main St., Brewer
Dwyer, Clement S.	205 French St., Bangor
Emery, Frederick C.	242 Cedar St., Bangor
Eyerer, Rudolf E.	489 State St., Bangor
Feeley, J. Robert	438 Garland St., Bangor
Fergus, Andrew	336 Mt. Hope Ave., Bangor
Gaillard, Richard A.	276 State St., Bangor
Gilman, Herbert C.	240 Penobscot Ave., Millinocket
Graves, Robert A.	Sunset Drive, Orono
Hall, Walter L. H.	130 Middle St., Old Town
Hamlin, Irving E.	Main St., E. Millinocket
Hill, Allison K.	431 State St., Bangor
Holzwarth, Hans A.	224 N. Main St., Brewer
Houlihan, John S.	209 State St., Bangor
Hughes, Edward J., Jr.	336 Mt. Hope Ave., Bangor
Hutchins, Deane L.	Health Dept., Univ. of Maine, Orono
Irwin, Carl W.	262 State St., Bangor
Kadi, Francis J.	Bangor State Hosp., Bangor
Kellogg, Robert O.	431 State St., Bangor
Leddy, Percy A.	Main St., Seal Harbor
Macdonald, Donald F.	263 State St., Bangor
Manter, Wilbur B.	1 Fern St., Bangor
Mason, Peter H.	Millinocket Com. Hosp., Millinocket
McEvoy, Charles D., Jr.	186 State St., Bangor
McGinn, John F.	205 French St., Bangor
McLean, Preston A.	336 Mt. Hope Ave., Bangor
McNamara, Wesley C.	8 Lee St., Lincoln
McQuoid, Robert M.	39 Columbia St., Bangor
Memmelaar, Joseph E.	431 State St., Bangor
Merriam, Thornton W., Jr.	431 State St., Bangor
Metcalf, Cecil J.	204 Parkway North, Brewer
Miragliuolo, Leonard G.	10 Maple St., Bangor
Moulton, Gardner N.	5 Grove St., Bangor
Munce, Richard T.	262 State St., Bangor
Nesin, Bourcard	10 Water St., Howland
Netland, Anders T.	431 State St., Bangor
O'Kane, Francis R.	122 Penobscot Ave., Millinocket
Osler, Jay K.	74 Birch St., Bangor
Palmer, Thomas H., Jr.	431 State St., Bangor
Parrot, Hadley	431 State St., Bangor
Pearson, John J.	100 S. Main St., Old Town
Pooler, Harold A.	Bangor State Hosp., Bangor
Porter, Edward C.	489 State St., Bangor
Purinton, William A.	276 State St., Bangor
Ridlon, Magnus F.	431 State St., Bangor
Ruhlin, Carl W.	336 Mt. Hope Ave., Bangor
Sewall, Elmer M.	14 Park St., Orono
Shapero, Benjamin L.	431 State St., Bangor
Shubert, Alice J.	317 State St., Bangor
Shubert, William M.	317 State St., Bangor
Shurman, Hans	10 Spring St., Dexter
Smith, Hugh A.	Eastern Maine Gen. Hosp., Bangor
Striar, Ronald R.	94 Essex St., Bangor
Strout, Warren G.	205 French St., Bangor
Sullivan, John R.	340 No. Main St., Brewer
Taylor, H. Lewis	25 Church St., Dexter
Thomas, Philip B.	205 French St., Bangor
Trowbridge, Mason, Jr.	142 Pine St., Bangor
Vickers, Martyn A.	268 State St., Bangor
Wadsworth, Richard C.	489 State St., Bangor
Wagner, Samuel L.	2 Holmes St., Winterport
Walker, George R.	336 Mt. Hope Ave., Bangor
Weisz, Hans	194 Main St., Lincoln
Whitney, Byron V.	280 State St., Bangor
Whitworth, John E.	336 Mt. Hope Ave., Bangor
Wood, George W., III	156 No. Main St., Brewer
Woodcock, John A.	35 Second St., Bangor

HONORARY

Craig, Allan	905 Egan Ave., Pacific Grove, Calif.
Devan, Thomas A.	325-19th Ave., N. E., St. Petersburg 4, Fla.

Hedin, Carl J.	Penobscot Terrace, Brewer
Higgins, George I.	15 Water St., Newport
McNeil, Harry D.	81 Silver Rd., Bangor
Weatherbee, George B.	Main St., Hampden Highlands
Woodcock, Allan	35 Second St., Bangor

SENIOR

Ames, Forrest B.	13 Longmeadow Dr., Brewer
Dunham, Rand A.	Box 400, East Millinocket
Emerson, W. Merritt	131 State St., Bangor
Merrill, Urban H.	Etna

SERVICE

Clough, Herbert T.
(Col.) USAF MC, 1086 Medical Service Sqdn., Randolph AFB, Texas 78148

PISCATAQUIS COUNTY

President — Norman H. Nickerson, M.D.
Secretary-Treasurer — Isaac Nelson, M.D.

ACTIVE

Bradbury, Francis W.	16 E. Main St., Dover-Foxcroft
Curtis, John B.	10 High St., Milo
Garcia-Rey, Felix M.	14 Charles St., Milo
Howard, George C.	Oak St., Guilford
Lightbody, Charles H.	No. Main St., Guilford
Nelson, Isaac	Box 336, Greenville
Nielsen, Odd S.	85 Pleasant St., Dexter
Rodriguez, Araminta M.	14 Charles St., Milo
Stitham, Linus J.	50 Main St., Dover-Foxcroft

HONORARY

Bundy, Harvey C.	Milo
MacDougal, Wilbur E.	186 Nowell Rd., Bangor
Winter address—Box 533, Bay Rd., Bonita Springs, Fla. 33923	
Pritham, Fred J.	Greenville Jct.

SENIOR

Nickerson, Norman H.	Greenville
Stanhope, Charles N.	South St., Dover-Foxcroft

JUNIOR

Johnson, James H., Jr.	Pine St., Centerville, Mass.

AFFILIATE

Carde, Albert M.	33 Elm St., Milo
Metcalf, John T.	47 Elm St., Milo

SOMERSET COUNTY

President — Marian L. Strickland, M.D.
Secretary-Treasurer — John P. Dow, M.D.

ACTIVE

Amrein, H. Carl	29 Weston Ave., Madison
Ball, Franklin P.	Bingham
Bernard, Albert J.	198 Madison Ave., Skowhegan
Briggs, Paul R.	Hartland
Dow, John P.	59 Main St., Pittsfield
Greenlaw, William A.	129 Main St., Fairfield
Grow, William B.	Central Maine San., Fairfield
Hoch, Gretl J.	Phillips
Hornstein, Louis S.	220 Water St., Skowhegan
Jordan, W. Edward, Jr.	68 Water St., Skowhegan
Kemezys, Kestutis M.	25 Garfield St., Madison
Laney, Richard P.	50 Water St., Skowhegan
McIntire, Percy C.	Central Maine San., Fairfield
Philbrick, Maurice S.	292 Water St., Skowhegan
Reed, Howard L.	68 Water St., Skowhegan
Smith, Edgar J.	1 Park St., Fairfield
Strickland, Marian L.	Easy St., Canaan
Sullivan, George E.	R.F.D. #1, Fairfield
Swett, Carlton E.	Fairview Hosp., Skowhegan
Sy, Vincente L.	Milford Ave., Bingham
Szelenyi, Ernest	Central Maine San., Fairfield
Szendey, Andrew M.	26 Gray St., Madison
Turner, Harland G.	Box 38, Norridgewock
Zbyczycki, Tadeo	39 High St., Skowhegan

HONORARY

Marston, Henry E.	No. Anson
Webber, Merlon A.	33 Lancey St., Pittsfield

SENIOR

Lord, Maurice E.	Box 537, Lake Placid, Fla.
Southworth, John D.	Hartland

WALDO COUNTY

President — John R. Webber, M.D.
Secretary-Treasurer — Seth H. Read, M.D.

ACTIVE

Albro, Ward A.	Northport Ave., Belfast
Caswell, John A.	16 Waldo Ave., Belfast
Cobb, Norman E.	132 Main St., Belfast
Read, Seth H.	15 Church St., Belfast
Stein, Ernest W.	72 Main St., Pittsfield
Temple, George L.	Fahey St., Belfast
Torrey, Raymond L.	Main St., Searsport
Webber, John R.	6 Northport Ave., Belfast

HONORARY

Small, Foster C.	169 High St., Belfast
Stevens, Carl H.	18 Franklin St., Belfast

WASHINGTON COUNTY

President — Robert G. MacBride, M.D.
Secretary-Treasurer — Karl V. Larson, M.D.

ACTIVE

Bates, James C.	Eastport
Brownrigg, Leslie W.	St. Stephen, N. B.
French, Rowland B.	16 Water St., Eastport
Gundersen, Peter M.	P. O. Box 279, Woodland
Jacob, Donald R.	Princeton
Kazutow, John	Box 113, Columbia Falls
Kiel, Joseph B.	Apt. 106, 2422 MacArthur Blvd., Irving, Texas 75060
Larson, Karl V.	E. Machias
MacBride, Robert G.	25 Washington St., Lubec
McAllister, John W.	39 Water St., Lubec
Mitchell, Hazen C.	Calais
Mundie, Perley J.	32 North St., Calais
Nackley, George N.	1 School St., Machias
Sears, Harold G.	Second Ave., Woodland

SENIOR

Bennet, DaCosta F.	4 Main St., Lubec

YORK COUNTY

President — Alvin A. Hoffman, M.D.
Secretary-Treasurer — Charles W. Kinghorn, M.D.

ACTIVE

Anton, Thomas	260 Main St., Biddeford
Bacon, Melvin	122 Main St., Sanford
Belmont, Ralph S.	6 Washington St., Sanford
Binette, Germain A.	331 Main St., Saco
Charest, Leandre R.	314 Alfred St., Biddeford
Cuneo, Kenneth J.	31 Summer St., Kennebunk
Dionne, William E.	75 Main St., Springvale
Dorfman, Irvin	11 Wildwood Dr., Saco
Downing, J. Robert	35 Summer St., Kennebunk
Drummond, S. Dunton	Bar Mills
Eisberg, Harry B.	331 Main St., Saco
Endicott, Ruth E.	Grasshopper Lane, Ogunquit
Ficker, Robert F.	Maine St., Kennebunkport
Fortier, Andre P.	68 Foss St., Biddeford
Haas, Carl M.	357 Elm St., Biddeford
Hazzard, Lawrence R.	Cider Hill Rd., York
Hill, Paul S., Jr.	323 Main St., Saco
Hoffman, Alvin A.	P.O. Box 38, York
Hopkins, Herbert J.	24 Portland Ave., Old Orchard Beach
Houle, Marcel P.	200 Alfred St., Biddeford
Jellerson, Leon R.	77 Franklin St., Boston 12, Mass.
Johnston, James S.	83 Main St., Kennebunk
LaFond, Robert S.	258 Main St., Saco
Lapirow, Harry	99 Main St., Kennebunk
Leigh, Kenneth E.	Brixham Rd., York
Lesieur, Louis C.	255 Beach St., Saco
Lincourt, Armand S.	122 Main St., Sanford
Lord, George A.	34 Winter St., Sanford
Lorentz, John J.	Maine Medical Ctr., Portland
Magaudda, Michael M. P.	39 Old Orchard St., Old Orchard Beach
Magocsi, Alexander W.	York
Moulton, Marion K.	W. Newfield
Murphy, John J.	84 Portland St., So. Berwick
Myer, John C.	Nasson College, Springvale
Nieuwkerk, Willem F.	P.O. Box 424, Kennebunkport
O'Sullivan, William B.	331 Main St., Saco
Ouellette, Marcel D.	120 Main St., Sanford
Patane, Joseph M.	256 Alfred St., Biddeford
Perrault, Oscar W.	30 South St., Biddeford
Peterlein, Walter R., Jr.	75 Main St., Springvale
Richards, Carl E.	34 Winter St., Sanford
Robert, Roger J. P.	331 Main St., Saco
Ross, Maurice	372 Main St., Saco
Roussin, William T.	48 Bacon St., Biddeford
Shaw, G. Patrick	357 Elm St., Biddeford
Smith, Gerald R.	Ogunquit
Smith, Oney P.	Post Rd., Wells
Taylor, Paul E.	9 Wentworth St., Kittery
Turville, Charles S.	P.O. Box 187, Alfred
Vachon, Robert D.	34 Winter St., Sanford
Viger, Leopold A.	10 Amherst St., Biddeford
Wolfahrt, Eugene P.	338 Main St., Saco

HONORARY

Bunker, Willard H.	York Harbor
Cobb, Stephen A.	34 Winter St., Sanford
Davis, Ansel S.	Springvale
Dennett, Carl G.	258 Main St., Saco
Kinghorn, Charles W.	4 Wentworth St., Kittery
Larochelle, Joseph R.	42 Bacon St., Biddeford
Ross, H. Danforth	34 Winter St., Sanford
Stevens, Harold W.	369 Ferry Rd., Saco
Whitney, Ray L.	Cape Porpoise

JUNIOR

Berger, Steven	Station B, Poughkeepsie, N. Y.

An Alphabetical List of the Members of the Maine Medical Association

The figures in parenthcses refer to County Societies as follows: (1) Androscoggin, (2) Aroostook, (3) Cumberland, (4) Franklin, (5) Hancock, (6) Kennebec, (7) Knox, (8) Lincoln-Sagadahoc, (9) Oxford, (10) Penobscot, (11) Piscataquis, (12) Somerset, (13) Waldo, (14) Washington, (15) York.

Abdalla, Abdalla Y., 19 Bedford St., Bath (8)
Adams, Asa C., 68 Main St., Orono (10)
Adams, Lester, 9 Knox St., Thomaston (9)
Adams, Marvin C., 25 Bramhall St., Portland (3)
Adams, Winford C., 588 North Main St., Brewer (10)
Agan, Robert W., 144 State St., Portland (3)
Akar, Hamdi, 17 Grove St., Bath (8)
Akerberg, Ake, 2 East Main St., South Paris (9)
Albert, Joseph L., 4 Pleasant St., Fort Kent (2)
Albert, Rodrigue J., 77 Main St., Fort Kent (2)
Albro, Ward A., Northport Ave., Belfast (13)
Alexander, Fay K., Camden Com. Hosp., Camden (7)
Allen, Donald E., Sebago Lake (3)
Ames, Forrest B., 13 Longmeadow Dr., Brewer (10)
Amfilo, Basil, 626 Main St., Lewiston (1)
Amrein, H. Carl, 29 Weston Ave., Madison (12)
Analis, Harry, 3 Forest Pk., Portland (3)
Anderson, Donald L., 369 Main St., Lewiston (1)
Anderson, Dorothy, 369 Main St., Lewiston (1)
Andrews, Anneliese M., Maine Medical Ctr., Portland (3)
Andrews, John F., 20 West St., Boothbay Harbor (8)
Ansell, Harvey B., 39 Deering St., Portland (3)
Anton, Thomas, 260 Main St., Biddeford (15)
Apollonio, Howard L., 22 White St., Rockland (7)
Applin, Hilton H., 6 Cumberland St., Brunswick (3)
Aranson, Albert, 39 Deering St., Portland (3)
Archambault, Philip L., 346 Main St., Lewiston (1)
Asali, Louis A., 29 Deering St., Portland (3)
Asherman, Edward G., 131 Chadwick St., Portland (3)
Ashley, Alta, 11 Weston St., Augusta (6)
Aucoin, Peter B., 151 Franklin St., Rumford (9)
Aungst, Melvin R., Morneault Building, Fort Kent (2)

Babalian, Leon, 645 Congress St., Portland (3)
Babcock, Edward B., 431 State St., Bangor (10)
Babcock, Harold S., Castine (5)
Bacastow, Merle S., 22 Bramhall St., Portland (3)
Bachrach, Louis, 16 Union St., Brunswick (3)
Bachulus, John M., P. O. Box 285, Bath (8)
Bacon, Melvin, 122 Main St., Sanford (15)
Baldini, Elio, 22 Bramhall St., Portland (3)
Baldwin, Warren C., 42 Deering St., Portland (3)
Ball, Franklin P., Bingham (12)
Barker, Nathaniel B. T., 1 South St., Yarmouth (3)
Barnard, John M. H., Doctors Park, 89 Hospital St., Augusta (6)
Barnes, Kirk K., Baribeau Dr., Brunswick (3)
Barrett, Robert J., Jr., Cor. Union & James Sts., Bangor (10)
Barron, Martin A., Jr., 37 Deering St., Portland (3)
Barron, Richard E., Main St., Monmouth (6)
Bates, James C., Eastport (14)
Bauman, Clair S., 159 Silver St., Waterville (6)
Bean, Achsa M., Star Route 22-282, Owl's Head (7)
Bean, H. Richard, 241 Main St., Norway (9)
Bearor, Robert A., Maine Medical Ctr., Portland (3)
Beaudet, Simon C., 25 Webster St., Lewiston (1)
Beckerman, Stanley C., 82 Elm St., Waterville (6)
Beeaker, Vincent H., 85 Wood St., Lewiston (1)
Beegel, Paul M., 80 Goff St., Auburn (1)
Beliveau, Bertrand A., 56 Howe St., Lewiston (1)
Belknap, Samuel L., Damariscotta (8)
Belmont, Ralph S., 6 Washington St., Sanford (15)
Bennet, DaCosta F., 4 Main St., Lubec (14)
Bennet, Eben T., 49 Deering St., Portland (3)
Berger, Steven, Station B, Poughkeepsie, N. Y. (15)
Bernard, Albert J., 198 Madison Ave., Skowhegan (12)
Bettle, Ronald A., 32 Federal St., Brunswick (3)
Betts, Anthony, Maine Medical Ctr., Portland (3)
Bhatnagar, Hemendra N., 30 Elm St., Waterville (6)
Bidwell, Robinson L., 31 Bramhall St., Portland (3)
Binette, Germain A., 331 Main St., Saco (15)
Bischoffberger, John M., Naples (3)
Bishop, Lloyd W., 211 Vaughan St., Portland (3)
Bjorn, John C., Hampden Highlands (10)
Black, Paul E., Capt., NAS, Lakehurst, N. J. (5)
Blackburn, Nelson P., Bath Memorial Hosp., Bath (8)
Blackwell, William M., Millinocket Com. Hosp., Millinocket (10)
Blaisdell, Carl E., 47 Broadway, Bangor (10)
Blaisdell, Elton R., 12 Deering St., Portland (3)
Blaisdell, William B., Jr., 47 Broadway, Bangor (10)
Blinder, Philip, 336 Mt. Hope Ave., Bangor (10)
Blumberg, Edward, 6316 Strickland Ave., Brooklyn 34, N. Y. (3)
Bolduc, Jean L., 173 Main St., Waterville (6)
Bonney, James H., 229 Vaughan St., Portland (3)
Boone, Storer W., 429 Main St., Presque Isle (2)
Bostwick, George W., P. O. Box 388, Newcastle (8)
Bourassa, Harvey J., 47 Elm St., Waterville (6)
Bove, Louis G., 12 Deering St., Portland (3)
Bowman, Peter W., P.O. Box C, Pownal (3)
Bowne, Hays G., 9A Main St., Farmington (4)
Boynton, Willard H., U.S. A.I.D., A.P.O. 271, New York, N. Y. (9)
Bradbury, Francis W., 16 E. Main St., Dover-Foxcroft (11)
Branch, Charles F., Central Maine Gen. Hosp., Lewiston (1)
Brann, Henry A., 31 Western Ave., Augusta (6)
Branson, Sidney R., 37 Main St., South Windham (3)
Breard, J. Alfred, 15 Summer St., Waterville (6)
Bridges, Donald E., 336 Mt. Hope Ave., Bangor (10)
Brien, Maurice, 76 Pine St., Lewiston (1)
Briggs, Paul R., Hartland (12)
Briggs, Winton, 507 Ocean St., South Portland (3)
Brinkman, Carl A., 50A Flaggy Meadow Rd., Gorham (3)
Brinkman, Harry, 47 Perham St., Farmington (4)
Brinkman, Paul A., Farmington (4)
Brito, Joseph S., St. Joseph Hosp., Bangor (10)
Brod, James J., 415 Broad St., Oneida, N. Y. 13421 (10)
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Brouwer, Johan, 5 Beech St., Rockland (7)
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Brown, Eugene E., 57 Summit Ave., Bangor (10)
Brown, Lloyd, 186 State St., Bangor (10)
Brown, Stephen S., Mars Hill (2)
Brownlow, Bradley E., Blue Hill Mem. Hosp., Blue Hill (5)
Brownrigg, Leslie W., St. Stephen, N. B. (14)
Buker, Edson B., R.F.D. No. 3, Auburn (1)
Bull, Frank B., Hasson St., Farmingdale (6)
Bullington, Sunny J., 32 Federal St., Brunswick (3)
Bundy, Harvey C., Milo (11)
Bunker, Willard H., York Harbor (15)
Burden, Charles E., 1 North St., Bath (8)
Burke, John E., 824 State St., Bangor (10)
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Burnham, Harold N., 130 Main St., Gorham (3)
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Chapin, Milan A., 237 Turner St., Auburn (1)

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Clark, Richard I., Freeport Medical Ctr., Freeport (3)
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Contartese, Michael, 149 Main St., Freeport (3)
Cook, Aaron, 23 High St., Waterville (6)
Cooper, Llewellyn W., 194 Main St., Bar Harbor (5)
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Coulton, Donald, 326 State St., Bangor (10)
Covert, Stanley B., Kingfield (4)
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Crawford, Albert S., East Blue Hill (6)
Crawford, J. Ramser, 105 Water St., Augusta (6)
Crawford, Joseph R., 105 Water St., Augusta (6)
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Crowe, James H., 57 Main St., Ellsworth (5)
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Cunningham, Alice N., 32 Federal St., Brunswick (3)
Curran, Edward L., 209 State St., Bangor (10)
Curtis, John B., 10 High St., Milo (11)
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Davidson, Gisela K., 235 State St., Portland (3)
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Davis, Ansel S., Springvale (15)
Davis, Earle M., 2 School St., Waterville (6)
Davis, Paul V., 94 Main St., Bridgton (3)
DeCosta, Donald A., Poland Spring (1)
Defoe, Garfield G., Dixfield (9)
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Dennis, Richard H., 33 College Ave., Waterville (6)
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Dionne, William E., 75 Main St., Springvale (15)
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Doble, Miriam, Sugarloaf Mt., Kingfield (8)
Doby, Tibor, 131 State St., Portland (3)
Dole, Richard R., 218 Main St., Waterville (6)
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Donahue, Gerald H., 4 Station St., Presque Isle (2)
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Dore, Clarence E., 2 School St., Waterville (6)
Dore, Kenneth E., 133 Main St., Fryeburg (3)
Dorfman, Irvin, 11 Wildwood Dr., Saco (15)
Dorogi, Louis V., Old Post Rd., Rt. 138, Bowdoinham (8)
Dougherty, John F., 112 Front St., Bath (8)
Douphinett, Otis J., 763 Congress St., Portland (3)
Dow, John P., 59 Main St., Pittsfield (12)
Downing, J. Robert, 35 Summer St., Kennebunk (15)
Drake, Emerson H., 18 Bramhall St., Portland (3)
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Drummond, S. Dunton, Bar Mills (15)
Duffey, Richard V., 187 North Main St., Brewer (10)
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Eddy, Robert H., 5 Beech St., Rockland (7)
Eisberg, Harry B., 331 Main St., Saco (15)
Elsemore, Dexter E., 11 Main St., Dixfield (9)
Emanuel, Meyer, Veterans Adm., Togus (6)
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Ferguson, Barbara, 80 Goff St., Auburn (1)
Ferguson, Franklin F., 22 Bramhall St., Portland (3)
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Ficker, Robert F., Maine St., Kennebunkport (15)
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Fiorica, Gaetano T., 12 Church St., Chisholm (4)
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Freeman, William E., 107 Main St., Yarmouth (3)
French, Rowland B., 16 Water St., Eastport (14)
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Frost, Robert A., 93 Summer St., Auburn (1)
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Giberson, Raymond G., 156 A Academy St., Presque Isle (2)
Giddings, Lane, 6 E. Chestnut St., Augusta (6)
Giddings, Paul D., 31 Western Ave., Augusta (6)
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Giguere. Eustache N., 90 Webster St., Lewiston (1)
Gilman, Herbert C., 240 Penobscot Ave., Millinocket (10)
Gingras, Adolphe J., 105 Water St., Augusta (6)
Gingras, Napoleon J., 6 East Chestnut St., Augusta (6)
Givertz, Bernard, 131 Chadwick St., Portland (3)
Glassmire, Charles R., 37 Deering St., Portland (3)
Godsoe, John A., 7 Bramhall St., Portland (3)
Goduti, Richard J., 9 Deering St., Portland (3)
Goldman, Morris E., 185 Webster St., Lewiston (1)
Good, Philip G., 38 Deering St., Portland (3)
Goodof, Irving I., Thayer Hospital, Waterville (6)
Goodrich, Blynn O., 165 Main St., Waterville (6)
Goodwin, Ralph A., Sr., 56 Denison St., Auburn (1)
Goodwin, Ralph A., Jr., 33 Court St., Auburn (1)
Gormley, Eugene G., Market Square, Houlton (2)
Gould, George I., 79 Main St., Richmond (6)
Graves, Robert A., Sunset Drive, Orono (10)
Gray, Philip L., Blue Hill (5)
Greco, Edward A., 12 Pine St., Portland (3)
Green, Ross W., 33 Court St., Auburn (1)
Greene, John P., 19 Sabattus St., Lewiston (1)
Greene, Merrill S. F., 466 Main St., Lewiston (1)
Greenlaw, William A., 129 Main St., Fairfield (12)
Gregory, Frederick J., So. Main St., Caribou (2)
Gregory, Philip O., St. Andrews Hosp., Boothbay Harbor (8)
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Griffiths, Eugene B., 350 Main St., Presque Isle (2)
Grimes, Gilbert R., 185 Webster St., Lewiston (1)
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Grow, William B., Central Maine San., Fairfield (12)
Guillemette, Maurice R., 109 Water St., Augusta (6)
Guite, L. Armand, Sr., 45 Elm St., Waterville (6)
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Haas, Rudolph, 480 Main St., Lewiston (1)
Hall, Walter D., 407 Main St., Rockland (7)
Hall, Walter L. H., 130 Middle St., Old Town (10)
Hallett, George W., Jr., 72 West St., Portland (3)
Hamilton, Virginia C., South Harpswell (8)
Hamlin, Irvin E., Main St., East Millinocket (10)
Handanos, Vassilios, Box C, Pownal (3)
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Hannigan, Margaret H., 85 Goff St., Auburn (1)
Harkins, Michael J., 437 Main St., Lewiston (1)
Harlow, Edwin W., 177 Main St., Waterville (6)
Harper, Harry L., 17 Main St., South Paris (9)
Harrison, George J., Market Sq., Houlton (2)
Harvey, Thomas G., 59 Mayo St., Caribou (2)
Harvey, William C., 59 Mayo St., Caribou (2)
Hawkes, Richard S., 47 Deering St., Portland (3)
Hawkins, Donald B., Atlantic Ave. and Sea St., Camden (7)
Hayward, I. Mead, So. Main St., Caribou (2)
Hazzard, Lawrence R., Cider Hill Rd., York (15)
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Herring, Leon D., Memorial Dr., Winthrop (6)
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Higgins, George I., 15 Water St., Newport (10)
Hill, Allison K., 431 State St., Bangor (10)
Hill, Douglas R., 855 Sawyer St., South Portland (3)
Hill, Frederick T., Thayer Hospital, Waterville (6)
Hill, Howard F., 33 College Ave., Waterville (6)
Hill, Kevin, 33 College Ave., Waterville (6)
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Hoch, Gretl J., Phillips (12)
Hoffman, Alvin A., P.O. Box 38, York (15)
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Holt, C. Lawrence, 27 Deering St., Portland (3)
Holz, Peter H., 51 Elm St., Camden (7)
Holzwarth, Hans A., 224 N. Main St., Brewer (10)
Hopkins, Herbert J., 24 Portland Ave., Old Orchard Beach (15)
Hornberger, H. Richard, 2 School St., Waterville (6)
Hornstein, Louis S., 220 Water St., Skowhegan (12)
Horsman, Donald H., 50 Goff St., Auburn (1)
Houle, Marcel P., 200 Alfred St., Biddeford (15)
Houlihan, John S., 209 State St., Bangor (10)
Howard, Emery B., Jr., 23A Summer St., Rockland (7)
Howard, George C., Oak St., Guilford (11)
Howard, Henry M., 105 Franklin St., Rumford (9)
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Larrabee, Charles F., 48 Mt. Desert St., Bar Harbor (5)
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Marshall, Joseph A., 177 Main St., Waterville (6)
Marshall, Paul A., 27½ Johnson Hgts., Waterville (6)
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Marston, Paul C., Kezar Falls (3)
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Metcalf, John T., 47 Elm St., Milo (11)
Michaud, Joseph C., 160 Silver St., Waterville (6)
Milazzo, John, 42 Elm St., Auburn (1)
Miller, Clark F., 46 Madison St., Auburn (1)
Miller, Hudson R., 11 Turner St., Auburn (1)
Miller, Thor, 752 Main St., Westbrook (3)
Milliken, Howard H., R #1, Pond Rd. (Manchester), Hallowell (6)
Millington, Paul A., 44 Mountain St., Camden (7)
Mills, Nathaniel, North Main St., Wolfeboro, N. H. (9)
Miragliuolo, Leonard G., 10 Maple St., Bangor (10)
Mitchell, Hazen C., Calais (14)
Mohlar, Robert G., Baribeau Dr., Brunswick (3)
Monaghan, Stephen E., 7 Bramhall St., Portland (3)
Monkhouse, William A., 131 State St., Portland (3)
Monsivais, Alfredo, 1 Western Ave., Winthrop (6)
Moore, Beryl M., High St., Oxford (9)
Moore, Valentine J., Thayer Hospital, Waterville (6)
Morin, Gerard L., 185 Webster St., Lewiston (1)
Morin, Harry F., 905 Middle St., Bath (8)
Morissette, Russell A., 185 Webster St., Lewiston (1)
Morris, Craig W., 50 Bangor St., Augusta (6)
Morrison, Alvin A., 57 Deering St., Portland (3)
Morrison, Robert M., 148 State St., Portland (3)
Morse, Edward K., 22 White St., Rockland (7)
Moulton, Albert W., Sr., 180 State St., Portland (3)
Moulton, Albert W., Jr., 180 State St., Portland (3)
Moulton, Gardner N., 5 Grove St., Bangor (10)
Moulton, Marion K., West Newfield (15)
Moulton, Sumner E., 12 Deering St., Portland (3)
Munce, Richard T., 262 State St., Bangor (10)
Mundie, Perley J., 32 North St., Calais (14)
Murphy, John J., 84 Portland St., South Berwick (15)
Myer, John C., Nasson College, Springvale (15)

McAdams, William R., 723 Congress St., Portland (3)
McAfee, Robert E., 18 Bramhall St., Portland (3)
McAllister, John W., 39 Water St., Lubec (14)
McCann, Eugene C., 49 Deering St., Portland (3)
McCrum, Philip H., 188 State St., Portland (3)
McEvoy, Charles D., Jr., 186 State St., Bangor (10)
McFarland, Edward A., Baribeau Dr., Brunswick (3)
McGinn, John F., 205 French St., Bangor (10)
McIntire, Barron F., Jr., 13 W. Elm St., Yarmouth (3)
McIntire, Percy C., Central Maine San., Fairfield (12)
McIntyre, John D., 50 Union St., Ellsworth (5)
McLaren, John J., Baribeau Dr., Brunswick (8)
McLarn, William D., 222 Main St., Waterville (6)
McLaughlin, Clarence R., 345 Water St., Gardiner (6)
McLaughlin, Ivan E., 345 Water St., Gardiner (6)
McLean, E. Allan, 29 Deering St., Portland (3)
McLean, Preston A., 336 Mt. Hope Ave., Bangor (10)
McLellan, William A., 87 Chestnut St., Camden (7)
McManamy, Eugene P., 72 West St., Portland (3)
McMichael, Morton, 73 Deering St., Portland (3)
McNamara, Wesley C., 8 Lee St., Lincoln (10)
McNeil, Harry D., 81 Silver Rd., Bangor (10)
McQuillan, Arthur H., 177 Main St., Waterville (6)
McQuoid, Robert M., 39 Columbia St., Bangor (10)

Nackley, George N., 1 School St., Machias (14)
Nadeau, J. Paul, 91 Pine St., Lewiston (1)
Nadeau, Lawrence A., 41 Sherbrooke Ave., Lewiston (1)
Nangle, Thomas P., West Paris (9)
Naumer, Harry A., Land's End, Port Clyde (7)
Nelson, Chesley W., 121 Main St., Norway (9)
Nelson, Isaac, Box 336, Greenville (11)
Nesin, Bourcard, 10 Water St., Howland (10)
Netland, Anders T., 431 State St., Bangor (10)
Nicholas, Eric F., Mars Hill (2)
Nichols, Arthur A., Edgecomb (8)
Nickerson, Norman H., Greenville (11)
Nielsen, Odd S., 85 Pleasant St., Dexter (11)
Nieuwkerk, Willem F., P.O. Box 424, Kennebunkport (15)
Nikolaidis, Demitrios, Augusta Gen. Hosp., Augusta (6)

O'Brien, William A., Arthur R. Gould Mem. Hosp., Presque Isle (2)
Oceretko, Arkadij, 37 Court St., Bath (8)
O'Connell, George B., 11 Lisbon St., Lewiston (1)
O'Connor, Francis J., 4 Woodlawn St., Augusta (6)
O'Donnell, Eugene E., 32 Deering St., Portland (3)
Oestrich, Alfred, 89 Congress St., Rumford (9)
Ohler, Robert L., Veterans Adm., Togus (6)
O'Kane, Francis R., 122 Penobscot Ave., Millinocket (10)
Olmsted, Burton L., 73 Deering St., Portland (3)
O'Meara, Edward S., 12 West Maple St., Ellsworth (5)
Onat, Mustafa V., St. George (7)
Orbeton, Everett A., 131 Chadwick St., Portland (3)
Osborne, John R., Narrows Pond Rd., Winthrop (2)
Osher, Harold L., 131 Chadwick St., Portland (3)
Osler, Jay K., 74 Birch St., Bangor (10)
O'Sullivan, William B., 331 Main St., Saco (15)
Ottum, Alvin E., 148 State St., Portland (3)
Ouellette, Benoit, 77 Main St., Fort Kent (2)
Ouellette, Marcel D., 120 Main St., Sanford (15)

Page, Rosario A., 22 Sweden St., Caribou (2)
Palmer, Thomas H., Jr., 431 State St., Bangor (10)
Parrot, Hadley, 431 State St., Bangor (10)
Patane, Joseph M., 256 Alfred St., Biddeford (15)
Patterson, James, 1 Bay Rd., South Portland (3)
Patterson, John C., Box 724, State Hospital, Augusta (6)
Pawle, Robert H., 8 Walcott Ave., Falmouth (3)
Pearson, John J., 100 So. Main St., Old Town (10)
Pease, Horace B., Maine Coast Mem. Hosp., Ellsworth (5)
Peddie, Harry M. K., Doctors Park, 89 Hospital St., Augusta (6)
Pena, Manuel G., 82 Maine Ave., Rumford (9)
Pendleton, Arthur D., 3 Green St., Fort Fairfield (2)
Pennoyer, Douglass C., 112 Vaughan St., Portland (3)
Penta, Walter E., 316 Woodford St., Portland (3)
Perkins, Niles L., 16 Bramhall St., Portland (3)
Perrault, Oscar W., 30 South St., Biddeford (15)
Peterlein, Walter R., Jr., 75 Main St., Springvale (15)
Petterson, Herman C., Chebeague Island (3)
Pfeiffer, Paul H., 14 Gilman St., Waterville (6)
Philbrick, Maurice S., 292 Water St., Skowhegan (12)
Phillips, David L., 82 Maine Ave., Rumford (9)
Pines, Philip, Maine St., Limestone (2)
Platt, Anna, Beauchamp Rd., Rockport (7)
Plimpton, Jay R., 283 Water St., Augusta (6)
Pogue, Jackson S., 529 Gilmore Ave., Trafford, Pa. 15085 (3)
Poliner, Irving J., 235 State St., Portland (3)
Polisner, Saul R., 143 Vaughan St., Portland (3)
Pomerleau, Ovid F., 179 Main St., Waterville (6)
Pomerleau, Rodolphe J. F., 27 Main St., Waterville (6)
Pooler, Harold A., State Hospital, Bangor (10)
Pope, W. Dean, 6 Pleasant St., Rangeley (4)
Porter, Edward C., 489 State St., Bangor (10)
Porter, Joseph E., 22 Bramhall St., Portland (3)
Potts, Ronald S., Central Maine Gen. Hosp., Lewiston (1)
Poulin, Albert A., Cherry Hill Dr., Waterville (6)
Poulin, James E., 177 Main St., Waterville (6)
Powell, Ralph C., Damariscotta (8)
Pratt, George L., 7 Main St., Farmington (4)
Pratt, Harold S., Livermore Falls (1)
Pratt, Loring W., 177 Main St., Waterville (6)
Price, Richard D., R.F.D. 2, Caribou (2)
Pritham, Fred J., Greenville Junction (11)
Proctor, Ray A., Garden Circle, Caribou (2)
Proctor, Thomas E., Boothbay Harbor (8)
Proudian, Paul O., 776 Main St., Westbrook (3)
Proulx, Harvey J., 92 Pine St., Lewiston (1)
Provost, Helen C., 48 Green St., Augusta (6)
Provost, Pierre E., 48 Green St., Augusta (6)
Purinton, William A., 276 State St., Bangor (10)

Rand, Carleton H., 219 Oak St., Lewiston (1)
Rando, Joseph J., 111 Webster St., Lewiston (1)
Ray, Ferris S., 7 Bramhall St., Portland (3)
Read, Seth H., 15 Church St., Belfast (13)
Reed, Howard L., 68 Water St., Skowhegan (12)
Reed, James W., 18 Main St., Farmington (4)
Reel, John J., 59 So. Front St., Richmond (6)
Reeves, Edward L., 179 Sabattus St., Lewiston (1)
Reeves, Helene M., 179 Sabattus St., Lewiston (1)
Reynolds, Arthur P., 29 Second St., Presque Isle (2)
Reynolds, John F., 216 Main St., Waterville (6)
Reynolds, Ralph L., 216 Main St., Waterville (6)
Richards, A. Dewey, 11 Gage St., Bridgton (3)
Richards, Carl E., 34 Winter St., Sanford (15)
Richards, Lee W., Jr., 21 Western Ave., Augusta (6)
Rideout, Samuel, Green St., Fort Fairfield (2)
Ridlon, Joseph R., 58 South St., Gorham (3)
Ridlon, Magnus F., 431 State St., Bangor (10)
Robert, Roger J. P., 331 Main St., Saco (15)
Robertson, George J., 171 Harrison Ave., Boston, Mass. (6)
Robinson, Hugh P., 27 Deering St., Portland (3)
Rock, Daniel A., 477 Main St., Lewiston (1)
Rodriguez, Araminta M., 14 Charles St., Milo (11)
Rodriguez, Jose M., 109 Silver St., Waterville (6)
Rogers, Albert M., 157 Pine St., Portland (3)
Rohm, Walter, Box 724, State Hosp., Augusta (6)
Root, John A., 22 White St., Rockland (7)
Rosenblatt, Stanley, 480 Main St., Lewiston (1)
Ross, H. Danforth, 34 Winter St., Sanford (15)
Ross, Maurice, 372 Main St., Saco (15)
Roussin, William T., 48 Bacon St., Biddeford (15)
Rowe, Gunther H., 42 Main St., Livermore Falls (4)
Rowe, Linwood M., Rumford Com. Hosp., Rumford (9)
Royal, Albert P., Jr., 82 Maine Ave., Rumford (9)
Rubins, Nina B., E. A. Center Mem. Clinic, Steep Falls (3)
Rubins, Talivaldis, E. A. Center Mem. Clinic, Steep Falls (3)
Ruhlin, Carl W., 336 Mt. Hope Ave., Bangor (10)
Runyon, William N., 283 Water St., Augusta (6)
Russell, Daniel F. D., Leeds (1)
Russell, Robert F., Penobscot (5)
Russell, Theodore M., 20 Western Ave., Augusta (6)

Sager, George F., 18 Bramhall St., Portland (3)
Sanders, Stephen W., 120 Main St., Winthrop (6)
Sanfacon, Philip G., Main St., Van Buren (2)
Sanford, Theodore H., 117 Goff St., Auburn (1)
Santoro, Domenico A., 43 Deering St., Portland (3)
Sapiro, Howard M., 171 State St., Portland (3)
Saunders, Allen I., Ferry Rd., R.F.D. 2, Augusta (6)
Saunders, Norman W., 12 Deering St., Portland (3)
Saunders, Sallie H., R.F.D., Camden (7)
Savage, Richard L., 4 Elm St., Fort Kent (2)
Sawyer, Howard P., Jr., 22 Bramhall St., Portland (3)
Schmidt, Lorrimer M., Veterans Adm., Togus (6)
Schumacher, William E., 14 Westwood Rd., MD "B," Augusta (6)
Schwarz, Harald J., Seton Hospital, Waterville (6)
Scolten, Adrian H., 32 Deering St., Portland (3)
Sears, Harold G., Second Ave., Woodland (14)
Seligman, Morris J., Veterans Adm., Togus (6)
Selvage, Irving L., Jr., 22 Bramhall St., Portland (3)
Senenky, Joseph P., Augusta State Hosp., Augusta (6)
Sewall, Elmer M., 14 Park St., Orono (10)
Sewall, Kenneth W., 2 School St., Waterville (6)
Shapero, Benjamin L., 431 State St., Bangor (10)
Shapiro, Morrill, 29 Deering St., Portland (3)
Shaw, G. Patrick, 357 Elm St., Biddeford (15)
Shaw, John H., 131 Sewall St., Augusta (6)
Shelton, M. Tieche, 21 Western Ave., Augusta (6)
Shems, Albert, 487 Main St., Lewiston (1)
Sherman, Fuller G., Spruce Pt., Boothbay Harbor (8)
Shields, Daniel R., 369 Main St., Lewiston (1)
Shubert, Alice J., 317 State St., Bangor (10)
Shubert, William M., 317 State St., Bangor (10)
Shurman, Hans, 10 Spring St., Dexter (10)
Sidwell-Thompson, Doris M., R.F.D. Whittier Rd., W. Ossipee, N. H. (3)
Simpson, Margaret R., 2 Sea Barn Rd., Cape Elizabeth (6)
Sirodot, George, Jackman (3)
Skillin, Charles E., 690 Congress St., Portland (3)
Skillin, Frederick W., 69 So. High St., Bridgton (3)
Sleeper, Francis H., 19 Columbia St., Augusta (6)
Small, Foster C., 169 High St., Belfast (13)
Smith, Carroll H., Box 785, Presque Isle (2)
Smith, Charles M., Main St., Dixfield (9)
Smith, Edgar J., 1 Park St., Fairfield (12)
Smith, Gerald R., Ogunquit (15)
Smith, Hugh A., Eastern Maine Gen. Hosp., Bangor (10)
Smith, Jacob, 118 Front St., Bath (8)
Smith, Joseph I., 118 Front St., Bath (8)
Smith, Kenneth E., Veterans Adm., Togus (6)

Smith, Margaret S., Box 967, Presque Isle (2)
Smith, Oney P., Post Rd., Wells (15)
Somerville, Robert B., 45 Hillside St., Presque Isle (2)
Somerville, Wallace B., Mars Hill (2)
Sommerfeld, Kurt A., Veterans Adm., Togus (6)
Soule, Gilmore W., 22 White St., Rockland (7)
Southern, Edward M., 1379 Balfour Rd., Grosse Pointe 30, Mich. (6)
Southworth, John D., Hartland (12)
Sowles, Horace K., 413 Blackstrap Rd., Falmouth (3)
Spear, William, 107 Main St., Lisbon Falls (1)
Spellman, Francis A., Veterans Adm., Togus (6)
Stadler, Eliot T., West Gouldsboro (5)
Stanhope, Charles N., South St., Dover-Foxcroft (11)
Stanwood, Harold W., c/o Kimball Stanwood, York (9)
Steele, Charles W., 472 Main St., Lewiston (1)
Stein, Ernest W., 72 Main St., Pittsfield (13)
Stephenson, Richard B., Bldg. 1, Rm. 118, National Institutes of Health, Bethesda, Md. 20014 (3)
Stetkevych, Alexander G., 858 Washington St., Bath (8)
Stetson, Rufus E., Damariscotta (8)
Stevens, Carl H., 18 Franklin St., Belfast (13)
Stevens, Harold W., 369 Ferry Rd., Saco (15)
Stevens, Theodore M., 148 State St., Portland (3)
Stimson, Barbara B., Star Route 22-282, Owl's Head (7)
Stinchfield, Allan J., P.O. Box 343, Augusta (6)
Stitham, Linus J., 50 Main St., Dover-Foxcroft (11)
Stocks, Joseph F., 67 Silver St., Waterville (6)
Storer, Daniel P., 108 Fessenden St., Portland (3)
Strach, Toffield B. J., 3 Deering St., Portland (3)
Striar, Ronald R., 94 Essex St., Bangor (10)
Strickland, Marian L., Easy St., Canaan (12)
Strout, Warren G., 205 French St., Bangor (10)
Sturtevant, Vaughn R., 33 College Ave., Waterville (6)
Sulkowitch, Hirsh, Box 1744, Portland (3)
Sullivan, George E., R.F.D. 1, Fairfield (12)
Sullivan, John R., 340 North Main St., Brewer (10)
Suyama, Eji, 58 W. Main St., Ellsworth (5)
Sweatt, Linwood A., 48 Drummond St., Auburn (1)
Swett, Alfred E., Maine Medical Ctr., Portland (1)
Swett, Carlton E., Fairview Hosp., Skowhegan (12)
Swett, Clyde I., 18 Sherman St., Island Falls (2)
Sy, Vincente L., Milford Ave., Bingham (12)
Sylvester, Stanley B., Union Mutual Life Ins. Co., 400 Congress St., Portland (3)
Szelenyi, Ernest, Central Maine San., Fairfield (12)
Szendey, Andrew M., 26 Gray St., Madison (12)

Tabachnick, Henry M., 110 Park Ave., Portland (3)
Tardif, Lionel R., 300 Pine St., Lewiston (1)
Taxiarchis, Louis N., 144 State St., Portland (3)
Taylor, H. Lewis, 25 Church St., Dexter (10)
Taylor, Paul E., 9 Wentworth St., Kittery (15)
Taylor, Richard W., St. Mary's Gen. Hosp., Lewiston (1)
Taylor, William F., 134 U.S. Route 1, Falmouth (3)
Tchao, Jou S., 82 Pine St., Lewiston (1)
Telfeian, Alphonse, 131 Chadwick St., Portland (3)
Temple, George L., Fahey St., Belfast (13)
Tetreau, William J., 144 Spring St., Portland (3)
Thacher, Henry C., 117 Goff St., Auburn (1)
Thaxter, Langdon T., Cumberland Foreside, Portland 200 (3)
Thegen, W. Edward, Elm St., Bucksport (5)
Thomas, Philip B., 205 French St., Bangor (10)
Thompson, Philip P., Jr., 131 Chadwick St., Portland (3)
Tibbetts, Otis B., 181 Gamage Ave., Auburn (1)
Titherington, John B., 97 Brook Rd., Falmouth (3)
Toner, Cornelius A., 31 Bramhall St., Portland (3)
Torrey, Marcus A., 75 State St., Ellsworth (5)
Torrey, Raymond L., Main St., Searsport (13)
Tougas, Raymond A., Baribeau Dr., Brunswick (3)
Tounge, Harry G., Jr., 12 Union St., Camden (7)
Tousignant, Camille, 111 Pine St., Lewiston (1)
Toussaint, Leonid G., P.O. Box 9, Fort Kent (2)
Towne, Charles E., 18 Common St., Waterville (6)
Tracy, Mary J., Bristol Rd., Damariscotta (8)
Trembly, Bruce, 33 College Ave., Waterville (6)
Trowbridge, Mason, Jr., 142 Pine St., Bangor (10)
Turcotte, Guy N., 7 Bramhall St., Portland (3)
Turcotte, Richard W., 70 Pine St., Lewiston (1)
Turgeon, Raphael F., 367 Main St., Westbrook (3)
Turnbull, Elliott D., Elm House, Naples (3)
Turner, Harland G., Box 38, Norridgewock (12)
Turville, Charles S., P.O. Box 187, Alfred (15)

Urjanis, Janis, Box C, Pownal (3)

Vachon, Robert D., 34 Winter St., Sanford (15)
Vanadzin, Boris A., 389 Congress St., Portland (3)
Van Deventer, Wilhelm H. J., 289 Maine St., Brunswick (3)
Van Lonkhuyzen, Maurice, 131 State St., Portland (3)
Veilleux, Lucien F., 30 Elm St., Waterville (6)
Vickers, Martyn A., 268 State St., Bangor (10)
Viger, Leopold A., 10 Amherst St., Biddeford (15)

Wadsworth, Richard C., 489 State St., Bangor (10)
Wagner, Samuel L., 2 Holmes St., Winterport (10)
Wakefield, Robert D., St. Mary's Hosp., Lewiston (1)
Walker, George R., 336 Mt. Hope Ave., Bangor (10)
Ward, John V., 131 State St., Portland (3)
Ward, William W., P.O. Box 804, Rockland (7)
Wasgatt, Wesley N., 41 Talbot Ave., Rockland (7)
Waterman, Richard, Waldoboro (7)
Weatherbee, George B., Main St., Hampden Highlands (10)
Weaver, Michael L., 36 Federal St., Brunswick (3)
Webber, Isaac M., 29 Deering St., Portland (3)
Webber, John R., 6 Northport Ave., Belfast (13)
Webber, Merlon A., 33 Lancey St., Pittsfield (12)
Webber, Wedgwood P., 299 Main St., Lewiston (1)
Weisz, Hans, 194 Main St., Lincoln (10)
Wellington, J. Foster, 396 Brighton Ave., Portland (3)
Westermeyer, Marion F., 25556 Lomas Verdes St., Loma Linda, Calif. 92354 (3)
Weymouth, Currier C., Eastmont Sq., Farmington (4)
White, Henry O., 22 White St., Rockland (7)
White, Leland M., So. Main St., Caribou (2)
White, William J., 1 Mitchell Rd., South Portland (3)
Whitney, Byron V., 280 State St., Bangor (10)
Whitney, Ray L., Cape Porpoise (15)
Whittier, Alice A. S., 143 Neal St., Portland (3)
Whitworth, John E., 336 Mt. Hope Ave., Bangor (10)
Wight, Donald G., 30 Mitchell Rd., South Portland (3)
Wilbur, Herbert T., Jr., 100 Main St., Southwest Harbor (5)
Willard, Harold N., Thayer Hospital, Waterville (6)
Williams, Edward P., 72 Main St., Houlton (2)
Williams, James A., 40 Pleasant St., Mechanic Falls (1)
Williamson, Elizabeth E., Blue Hill (5)
Williamson, Russell G., Blue Hill Mem. Hosp., Blue Hill (5)
Willie, James A., 111 Webster St., Lewiston (1)
Wilson, G. Ivan, 40 Court St., Houlton (2)
Wilson, Harry M., 944 Middle St., Bath (8)
Wilson, Robert D., Mt. Desert Island Hosp., Bar Harbor (5)
Wilson, Robert W., Veterans Adm., Togus (6)
Winchenbach, Francis A., 910 Washington St., Bath (8)
Winkelbauer, Rudolf G., Baribeau Dr., Brunswick (3)
Wolfahrt, Eugene P., 338 Main St., Saco (15)
Wood, George W., III, 156 North Main St., Brewer (10)
Woodcock, Allan, 35 Second St., Bangor (10)
Woodcock, John A., 35 Second St., Bangor (10)
Woodman, Arthur B., 15 Johnson Rd., Falmouth Foreside (3)
Worthing, Verla E., Box A, Thomaston (7)
Wyman, David S., 47 Deering St., Portland (3)

York, Elihu, U.S.N. Air Development Ctr., AMAL, Johnsville, Pa. 18974 (5)
Young, E. Stanley, Poland Spring (1)
Young, John, Paradise St., Bethel (9)

Zanca, Ralph, 86 Pine St., Lewiston (1)
Zbyczycki, Tadeo, 39 High St., Skowhegan (12)
Zeller, Alan W., 35 Main St., Damariscotta (8)
Zikel, Herbert M., High St., Wilton (4)
Zolov, Benjamin, 296 Congress St., Portland (3)

Past Presidents

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*Joseph H. Estabrook, M.D., Camden	1855-1856
*Hosea Rich, M.D., Bangor	1856-1857
*Gilman Daveis, M.D., Portland	1857-1858
*J. C. Bradbury, M.D., Old Town	1858-1859
*H. H. Hill, M.D., Augusta	1859-1860
*T. G. Stockbridge, M.D., Bath	1860-1861
*H. M. Harlow, M.D., Augusta	1861-1862
*Alonzo Garcelon, M.D., Lewiston	1862-1863
*J. T. Gilman, M.D., Portland	1863-1864
*N. P. Monroe, M.D., Belfast	1864-1865
*Amos Nourse, M.D., Bath	1865-1866
*S. H. Tewksbury, M.D., Portland	1866-1867
*Cyrus Briggs, M.D., Augusta	1867-1868
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*D. McRuer, M.D., Bangor	1869-1870
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*A. J. Fuller, M.D., Bath	1871-1872
*A. P. Snow, M.D., Winthrop	1872-1873
*A. F. Page, M.D., Bucksport	1873-1874
*Thomas H. Brown, M.D., Paris	1874-1875
*J. H. Bates, M.D., Yarmouth	1875-1876
*E. F. Sanger, M.D., Bangor	1876-1877
*T. H. Jewett, M.D., South Berwick	1877-1878
*M. C. Wedgwood, M.D., Lewiston	1878-1879
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*William Warren Greene, M.D., Portland	1880-1881
*A. K. P. Meserve, M.D., Buxton	1881-1882
*George E. Brickett, M.D., Augusta	1882-1883
*Oren A. Horr, M.D., Lewiston	1883-1884
*Thomas A. Foster, M.D., Portland	1884-1885
*Sumner Laughton, M.D., Bangor	1885-1886
*J. B. Walker, M.D., Thomaston	1886-1887
*Frederick C. Thayer, M.D., Waterville	1887-1888
*Stephen H. Weeks, M.D., Portland	1888-1889
*Benjamin F. Sturgis, M.D., Auburn	1889-1890
*Samuel B. Hunter, M.D., Machias	1890-1891
*Edwin M. Fuller, M.D., Bath	1891-1892
*Alfred Mitchell, M.D., Brunswick	1892-1893
*John A. Donovan, M.D., Lewiston	1893-1894
*W. P. Giddings, M.D., Gardiner	1894-1895
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*D. A. Robinson, M.D., Bangor	1896-1897
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*Charles O. Hunt, M.D., Portland	1898-1899
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*Edward H. Hill, M.D., Lewiston	1900-1901
*Frederic H. Gerrish, M.D., Portland	1901-1902
*Hiram Hunt, M.D., Greenville	1902-1903
*Augustus S. Thayer, M.D., Portland	1903-1904
*F. L. Dixon, M.D., Lewiston	1904-1905
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*C. E. Williams, M.D., Auburn	1906-1907
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*Alfred D. Sawyer, M.D., Fort Fairfield	1908-1909
*Galen M. Woodcock, M.D., Bangor	1909-1910
*E. H. Bennett, M.D., Lubec	1910-1911
*Stanley P. Warren, M.D., Portland	1911-1912
*Ralph H. Marsh, M.D., Guilford	1912-1913
*W. C. Peters, M.D., Bangor	1913-1914
*H. L. Bartlett, M.D., Norway	1914-1915
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*George H. Coombs, M.D., Waldoboro	1918-1919
*H. B. Mason, M.D., Calais	1919-1920
*Theodore E. Hardy, M.D., Waterville	1920-1921
*Addison S. Thayer, M.D., Portland	1921-1922
*L. T. Snipe, M.D., Bath	1922-1923
*C. A. Moulton, M.D., Hartland	1923-1924
*F. W. Mann, M.D., Houlton	1924-1925
*J. D. Phillips, M.D., Southwest Harbor	1925-1926
*L. P. Gerrish, M.D., Lisbon Falls	1926-1927
*Herbert F. Twitchell, M.D., Portland	1927-1928
*Frank Y. Gilbert, M.D., Portland	1928-1929
*Delbert M. Stewart, M.D., South Paris	1929-1930
*Charles B. Sylvester, M.D., Portland	1930-1931
*Ernest V. Call, M.D., Lewiston	1931-1932
*E. Delmont Merrill, M.D., Dover-Foxcroft	1932-1933
Warren E. Kershner, M.D., Bath	1933-1934
*Edwin W. Gehring, M.D., Portland	1934-1935
*John L. Johnson, M.D., Bangor	1935-1936
Frederick T. Hill, M.D., Waterville	1936-1937
*Ralph W. Wakefield, M.D., Bar Harbor	1937-1938
Willard H. Bunker, M.D., York Harbor	1938-1939
George L. Pratt, M.D., Farmington	1939-1940
Thomas A. Foster, M.D., Portland	1940-1941
*P. L. B. Ebbett, M.D., Houlton	1941-1942
Carl H. Stevens, M.D., Belfast	1942-1943
*Oscar F. Larson, M.D., Machias	1943-1944
*R. V. N. Bliss, M.D., Blue Hill	1944-1945
*Adam P. Leighton, M.D., Portland	1945-1946
*John O. Piper, M.D., Waterville	1946-1947
Stephen A. Cobb, M.D., Sanford	1947-1948
Forrest B. Ames, M.D., Bangor	1948-1949
Ralph A. Goodwin, M.D., Auburn	1949-1950
Foster C. Small, M.D., Belfast	1950-1951
C. Harold Jameson, M.D., Rockland	1951-1952
*Eugene H. Drake, M.D., Portland	1952-1953
Norman H. Nickerson, M.D., Greenville	1953-1954
*Robert W. Belknap, M.D., Damariscotta	June-August 1954 (Died in Office)
*William F. Mahaney, M.D., Saco	1954-1955
Martyn A. Vickers, M.D., Bangor	1955-1956
*Armand Albert, M.D., Van Buren	1956-1957
Francis A. Winchenbach, M.D., Bath	1957-1958
Eugene E. O'Donnell, M.D., Portland	1958-1959
Allan Woodcock, M.D., Bangor	1959-1960
*Wilson H. McWethy, M.D., Augusta	June 1960-February 1961 (Died in Office)
Carl E. Richards, M.D., Sanford	February 1961-June 1961
James A. MacDougall, M.D., Rumford	1961-1962
*Ralph C. Stuart, M.D., Guilford	1962-1963
Ernest W. Stein, M.D., Pittsfield	1963-1964
Thomas A. Martin, M.D., Portland	1964-1965

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* Deceased

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